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Monthly Message

Each spring there is a NATO Military Medical Conference which lasts three days. The 1958 meeting, arranged by Major General William F. Powell, USAF (MC), Chief of the Medical Section, was held in Paris on April 1, 2, and 3. As an innovation this year the three days were divided, with the first at Headquarters SHAPE, the second at the U. S. Air Base at Dreux, and the third at the French School of Aviation Medicine at Bretigny.

At the first session there was an address by General Norstad and briefings by his staff. In the afternoon the Norwegian group gave an excellent presentation of teaching methods including films and demonstrations by personnel, some so realistic that several in the audience thought they were true occurrences.

The meeting on the second day, the British, under the direction of Lieutenant General Sir Alexander Drummond, RAMC, brought in a 100-bed surgical hospital by air and had it completely established, equipped, and working with simulated patients within three hours. The group saw the arrival by air, the unloading of the airplanes, and then three hours later saw the hospital in full operation even to the digging of latrine pits. In the afternoon the group inspected various types of U. S. transport, cargo, and medical evacuation aircraft on the base, witnessed the U. S. Navy demonstration, and saw a Turkish film of evacuation.

The third day was spent at Bretigny at the French School of Aviation Medicine, with demonstrations by the group.

We returned to Paris and were entertained at the Val de Grace for lunch and during the afternoon. This has been the official military hospital of France since the days of Napoleon. The buildings date back about a century and a half to the first half of the 17th century, when they were originally built as a convent.

Over one hundred delegates and guests were in attendance.

Frank B. Berry

FRANK B. BERRY, M. D.
Assistant Secretary of Defense
(Health and Medical)

Table of Contents

Page

Human Tolerance to Some of the Accelerations Anticipated in Space Flight—*Stuart Bondurant, Neville P. Clarke, William G. Blanchard, Hugh Miller, Rufus R. Hessberg, Jr., and Edwin P. Hiatt* 1093

Heat Stroke—*Jerome Fabricant* 1106

Extracorporeal Circulation for Open Heart Surgery in a Naval Hospital—*Joseph J. Timmes, Robert D. Sellers, Marvin L. Gliedman, Romulus L. May, Robert J. Fleischaker, Daniel M. Pino, Sarkis S. Sarkisian, York Jacobson, and Karl E. Karlson* 1119

Exchange Transfusion for Aucte Poisoning in Children—*Ogden C. Bruton* 1128

Pulmonary Vascular Changes With Stenosis of Pulmonary and Tricuspid Valves and Small Right Ventricle—*Edward A. Smuckler, and Robert C. Hastedt* 1132

The Role of Elective Plastic Surgery in Military Hospitals—*Thomas D. Rees, William C. Trier, and Clarence R. Straatsma* 1146

The Management of Late Problems Following Pediatric Cardiovascular Surgery—*Bernard A. Yablin and Nicholas G. Bottiglieri* 1155

Child Guidance Clinic: Principles and Criteria for Establishment and Evaluation—*Frederick A. Zehrer* 1161

SERVICE ARTICLE

Martin Army Hospital, Fort Benning, Georgia—*James P. Cooney* 1168

CLINICOPATHOLOGIC CONFERENCE

U. S. Naval Hospital, St. Albans, N. Y. 1176

CASE REPORTS

Speculations Concerning the Influence of Psychoneurosis on Cardiac Pain—*Robert Spencer* 1184

Constitutional Hepatic Dysfunction in a Family—*Charles M. Weber* 1198

Left-Sided Traumatic Diaphragmatic Hernia Involving Solid Viscera—*C. William Rogers* 1204

Juvenile Fibromatosis—*Theodore E. Waugh* 1208

Ureteral Ectopia—*Anthony A. Borski and Robert W. Parsons* 1213

DEPARTMENTS

New Martin Army Hospital Dedicated in Ceremony at Fort Benning, Georgia 1217

Admiral Hogan Given Private Audience With Pope Pius XII on European Tour 1218

Navy Medical Reserve Consultants Board Discusses Graduate Training Program 1219

DEPARTMENTS—Continued

	Page
A Message From the A. M. A.	1220
World Congress of Aviation Medicine	1221
Second Army Consultants and Hospital Commanders Meet at Fort George Meade	1222
West Point Hospital Given Accreditation	1223
Official Decorations	1223
Army Dedicates New Hospital on Okinawa	1224
Captain Gorby Receives Hoff Medal	1224
Officers Certified by Specialty Boards	1225
Dr. Youmans Named Medical Research Advisor	1226
Col. Snyder Heads Physical Therapists	1226
Deaths	1227
Army Chemical Corps Develops New Mask	1227
Reviews of Recent Books	1228
New Books Received	1243

Foreword

The *United States Armed Forces Medical Journal* is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense. The Assistant Secretary of Defense (Health and Medical) and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this *Journal.*

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UNITED STATES ARMED FORCES MEDICAL JOURNAL

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HUMAN TOLERANCE TO SOME OF THE ACCELERATIONS ANTICIPATED IN SPACE FLIGHT

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RECENT technical advances have made human space travel imminent. The design of space vehicles and propulsion systems requires a comprehensive source of information concerning human tolerance limits to the accelerations which are expected. It is the purpose of this report to present a summary of the best current knowledge of these tolerance limits. This summary is based on published data and on studies which will be published shortly. For important details and comprehensive evaluation of specific points, the individual reports should be consulted.*

This report will treat only those linear accelerations which are expected during exit and re-entry. It will not treat the zero g of orbital flight, nor will it cover the brief forces which may be generated in an emergency escape from the space vehicle or on impact landing.

From Aero Medical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio. WADC Technical Report 58-156.

*In this report, the actual direction of acceleration is generally indicated. The magnitude of this acceleration is expressed in "g" units. The conventional terms "positive G", "negative G", and "transverse G" are occasionally used to indicate the physiologically important inertial force which is equal in magnitude but opposite in direction to the force causing acceleration. This force is expressed in "G" units.

METHODS

All of the data presented here are based on determinations made on human centrifuges. The use of the centrifuge to produce acceleration introduces the artifact of angular acceleration of larger magnitude than that expected during exit and re-entry. The possible significance of this artifact is treated in the discussion which follows.

Subjects in all tests were healthy young males, experienced in riding the centrifuge. Rates of onset of acceleration were varied as indicated between 0.1 g and 8 g per second.

In most of the studies a "plateau" acceleration pattern has been used.[1-8,10] The acceleration was increased to the predetermined level which was maintained until a physiologic or an arbitrary end point was reached. The typical rocket profiles were not followed in these experiments, because preliminary observations revealed no evidence of marked decrease in tolerance resulting from intermittent breaks in the acceleration pattern. The data should therefore be applicable to many anticipated acceleration patterns.

In some of the studies, seated subjects have been exposed to forward acceleration patterns similar to those produced by a three-stage rocket system.[3] These patterns consisted of successive accelerations to three instantaneous peaks of either 8, 10, or 12 g. After each peak, deceleration was effected as rapidly as possible (20 to 35 seconds) to 1.5 g and the next acceleration begun immediately. The rate of increase of acceleration was constant throughout each run. It was selected so that if acceleration were stopped abruptly at the peak of each stage (as with most rockets), the total acceleration would be sufficient to increase velocity by more than 18,000 miles per hour. Because the centrifuge could not be stopped abruptly, the subjects actually tolerated 15 to 25 percent more acceleration than required with an equivalent rocket pattern. In the series with three peaks at 12 g, the rate of increase of acceleration was 1 g per 4.5 seconds; in the series with three peaks at 10 g, the rate was 1 g per 7 seconds; and in the series with three peaks at 8 g, the rate was 1 g per 12 seconds.

The duration of tolerance to accelerations of 6, 8, 10, and 12 g has been studied in semisupine subjects immersed in a tank of water on the centrifuge.[2] Respiration was maintained under these conditions by delivering air for ventilation at a pressure equal to the hydrostatic pressure acting on the chest wall. This pressure was regulated by a skin diver's valve mounted under water at chest level.

The term "tolerance limit," as used in these studies, indicates the point at which volunteer subjects were unable to withstand continued acceleration. The subjects were instructed to termi-

nate the acceleration when vision became seriously impaired (loss of peripheral lights), when respiration became impossible, or when pain reached an intensity which was estimated to be sufficient to impair judgment or performance. Experience with the centrifuge and motivation were thus unquestionably among the factors which determined the tolerance limits reported here. Within the stated tolerance limits, the subject would be expected to see, think, and exercise at least finger control. The accuracy of coordination and competency of judgment under these conditions remain to be evaluated.

DATA

The curves of figure 1 are intended to approximate tolerance limits for 50 per cent of a young male population. The orbital velocity curve represents the time required at each g level to reach these velocities. This curve is included for comparison with the tolerance curves.

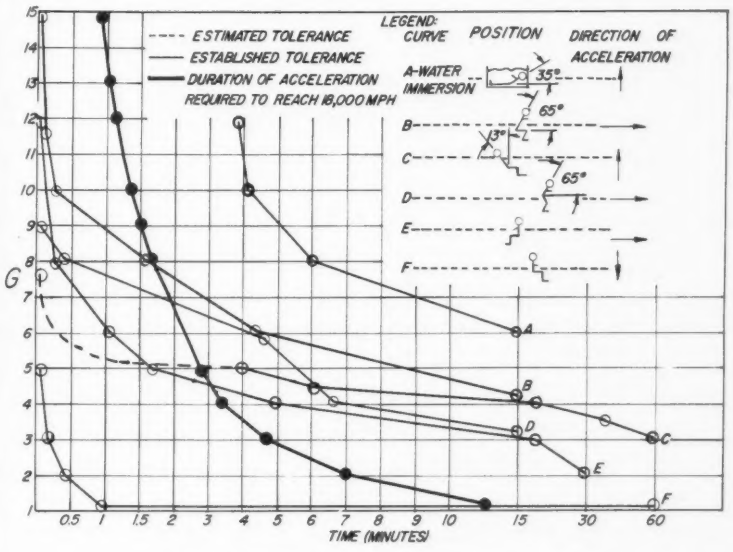

Figure 1. The duration of tolerance to acceleration.

Certain considerations are of importance in determining the location of each of the tolerance curves.

Transverse G

In those positions (A, B, and D) in which the subject faces in the direction of acceleration, the angle of the trunk, relative to the direction of acceleration, is critical. Tolerance to forward acceleration in the conventional seated position (fig. 2,

B2) is limited at 8 g by dyspnea (difficult breathing) and chest pain. In addition, there is a component of negative G (footward acceleration) resulting from the backward tilt of the trunk. If the angle of the trunk, relative to the direction of acceleration, is greater than 70° (fig. 2, B1), a severe, quasi-pleuritic anterior chest pain limits tolerance at about 7 g. As the angle is de-

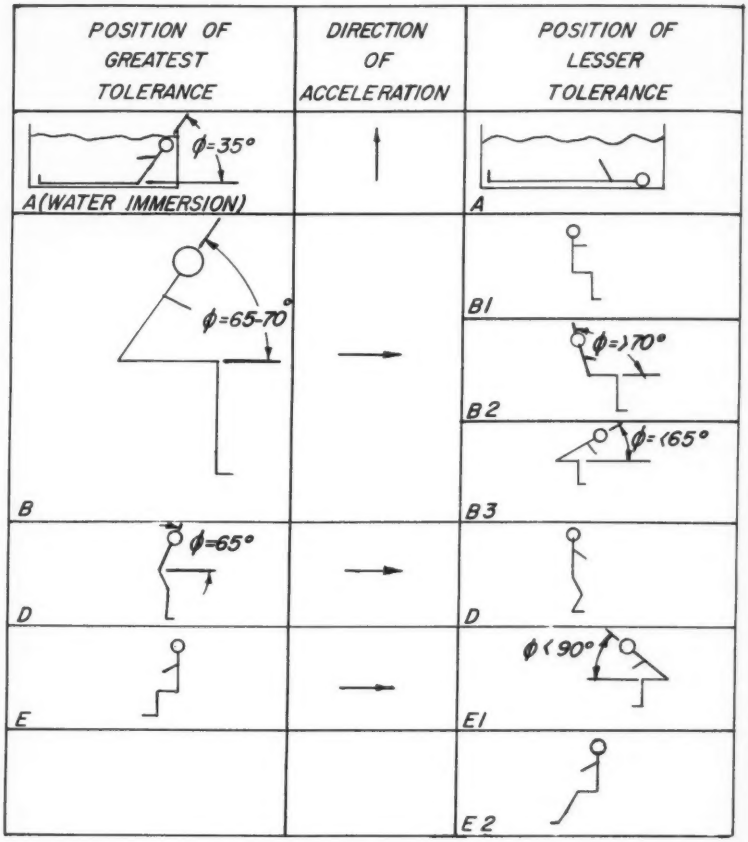

Figure 2. Variations in position which decrease tolerance to acceleration (see text).

creased below 70°, (fig. 2, B3), there is a more longitudinal application of the inertial force (positive G), and blackout limits tolerance at progressively lower g levels, (fig. 2, B3). The best tolerances have been obtained with the subject leaning in the direction of acceleration at a 65° to 70° angle. Above 8 g, blackout limits tolerance time in positions B (legs flexed) and D (semisupine). A greater tolerance in position B (12 g) than in

position D (10 g) was demonstrated by comparing the tolerance of the same subjects in the two positions (fig. 1). This is assumed to be due to a greater displacement of blood from the legs toward the trunk in position B, an effect like that caused by an anti-G suit. The tolerances in position D which are reported here are less than those reported previously.[1] The values obtained in the more recent study are used so that the relative merit of the two positions can be compared. The portions of these curves at and below 12 g are from work soon to be published.[3] The portions above 12 g are from other sources.[4, 5]

In positions B and D, respiration becomes difficult above 4 g. Inability to maintain adequate respiration is the most common limiting factor at 6 and 8 g. Thoracic breathing is almost impossible at these g levels, and tolerance time is largely determined by the ability of the subject to practice abdominal breathing.

Above 6 g, petechiae of the back and the antecubital fossae occur consistently. The petechiae are usually not painful, do not interfere with performance, and are absorbed in two or three days.

In these positions (B and D), the subject is supported by the seat. Minimal restraint harness is necessary.

Disability after acceleration in these positions is variable. All subjects could make pertinent observations of the effect of the acceleration immediately after the run. Most were able to perform coordinated hand and arm movements before the centrifuge stopped. The accuracy of these movements was not tested. All were able to walk with an unsteady gait within a minute after acceleration. The unsteady gait, along with dizziness, vertigo, and occasionally, nausea persisted for one to five minutes after the run.

Tolerance of the seated, forward-facing subject to backward acceleration (equivalent to the deceleration of re-entry) is indicated in figure 1, curve E, and in figure 3. Two factors are of importance in determining this tolerance limit.

First, the restraint system must be one which distributes the force evenly over large areas of the body without pressure points or local vascular occlusions. The tolerances indicated here were obtained with a suit designed to meet these requirements. Current operational harnesses are not adequate. A maximum of 5 g was tolerated with several harnesses basically similar to the U. S. Air Force integrated harness.

Second, the position of the body must be as indicated in figure 1, E. If the head is allowed to move forward during acceleration (fig. 2, E1), an effective negative G component is introduced. The greater the forward movement of the head, the lower is the

tolerance. If the legs are extended (fig. 2, E2), intense calf and thigh pain limits tolerance at about 5 g. This pain may persist for several days. It is assumed to be related to the local vascular distension and high pressures which occur with backward acceleration in this position. Wrapping the legs with elastic bandages and voluntary tensing of the leg muscles ameliorate the pain somewhat, but these technics have not measurably improved tolerance in position E2.

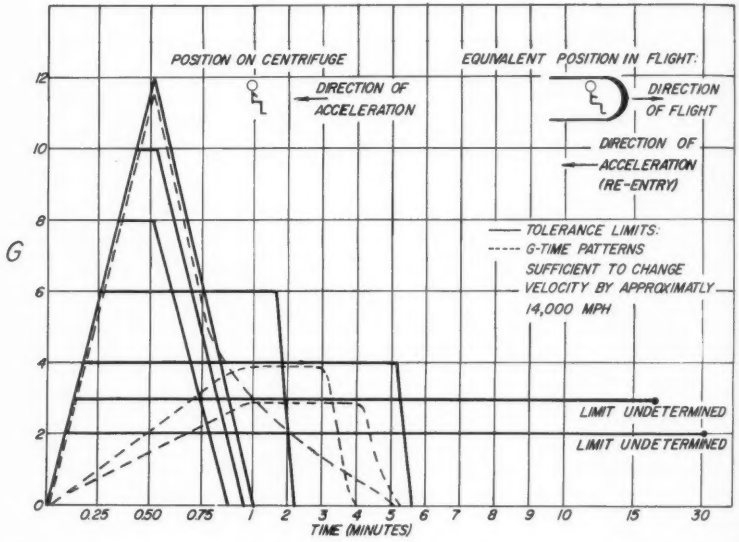

Figure 3. Tolerance of seated, forward-facing subject to deceleration in line of flight (re-entry).

In the optimal position for backward acceleration (fig. 1, E), tolerance to accelerations of 4 to 8 g is generally limited by leg pain similar to that described above. At levels above 8 g, both leg pain and dyspnea have been limiting factors. Petechiae occur consistently over the hands and anterior legs at high g levels.

Residual effects of acceleration in position E have been essentially like those described above for positions B and D.[3]

Positive G

The seat used in the studies of positive G tolerance has a 13° backward tilt of the backrest. The subjects sat with legs partially extended.

Headward acceleration can be tolerated by the subject in this position without an anti-G suit for the times indicated in

figure 1, curve C, if the rate of acceleration to each g level is approximately 0.1 g per second. At higher rates of acceleration, an anti-G suit may be required by some subjects. Since the subject is supported by the seat, minimal restraint harness is necessary. Petechiae occur commonly over the legs, feet, and lower arms after more than 10 minutes above 3.5 g. Tolerance in this position is generally limited below 4 g by fatigue, backache, and headache.

Because of the slow rate of onset of acceleration, blackout is rare below 4 g. It is the limiting factor in many subjects at 4 to 5 g. Occasional episodes of profuse sweating, pallor, nausea, tachycardia, and a sensation of imminent loss of consciousness have occurred in this position without relation to the magnitude or duration of the acceleration. Acceleration was stopped immediately in each instance. Weakness, dizziness, malaise, and nausea often persisted for several hours after these episodes.

Residual effects after headward acceleration for the times indicated in figure 1, C, are highly variable among subjects, and for the same subject on different days. Residual effects may be as slight as those described for positions B and D above, or weakness, dizziness, and malaise may be prostrating for several hours.

Negative G

The tolerance curve for footward acceleration refers to the seated subject (fig. 1, F). The upright subject would probably be less tolerant of negative G. Protective devices (pressure helmets) can be used to increase this tolerance limit. Even so, this is the position of least tolerance. Tolerance time is generally limited by head and eye pain. Subconjunctival hemorrhages and transient cardiac arrest have been reported.[10]

Water Immersion

Water immersion (fig. 1, A) allows tolerances at least as great as those indicated. The angle of the trunk, relative to the direction of acceleration, is critical whether the subject is in water or in air. The 35° angle indicated in position A, figure 1, seems to be optimal for the immersed subject. Restraint of the immersed subject is unnecessary. Petechiae do not occur during acceleration to 12 g with the subject immersed. Among the advantages of water immersion is the freedom of movement of the subject regardless of the g level. The freedom with which the head can can be moved at high g levels makes labyrinthine stimulation likely and vertigo a potential problem. If the head is held in one position, dizziness is only moderate, persisting for one to five minutes after acceleration.[2]

Rocket-like Acceleration Patterns

Figure 4 presents three three-stage rocket-like acceleration patterns which have been tolerated by a small group of subjects in position B. The residual symptoms after these accelerations were highly variable. All subjects were able to talk and to move arms and legs before the centrifuge stopped. Crude tests of coordination of the hands and arms were performed within one minute after acceleration. No marked deficit was observed. Some subjects experienced marked malaise and dizziness for five minutes to one hour after acceleration. In this small series, these symptoms were observed mostly in inexperienced subjects.[2]

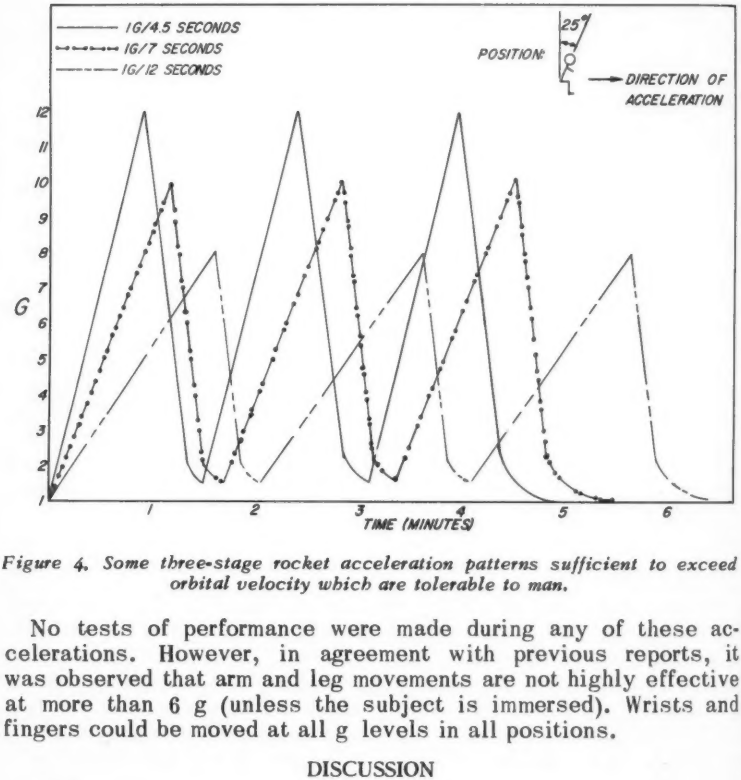

Figure 4. Some three-stage rocket acceleration patterns sufficient to exceed orbital velocity which are tolerable to man.

No tests of performance were made during any of these accelerations. However, in agreement with previous reports, it was observed that arm and leg movements are not highly effective at more than 6 g (unless the subject is immersed). Wrists and fingers could be moved at all g levels in all positions.

DISCUSSION

Exit Acceleration Patterns

Acceleration of the magnitudes and durations anticipated during exit from the earth's atmosphere can be tolerated without anti-G equipment in at least two positions (B and D). Of these two positions, the former (position B, seated) has two advantages:

tolerance is greater by approximately 2 g, as tested on the same subjects;-and a smaller top-to-bottom cabin dimension is required to accommodate the subject. This position has the disadvantage of being rather uncomfortable for long periods of time when there is no forward acceleration. An adjustable seat back would be required to enable the necessary forward tilt during forward acceleration and the more comfortable upright position during flight. Deceleration would be poorly tolerated by the subject in positions B or D, because the forward tilt would introduce a negative G component. For this reason, in either position there should be provision for immediate return to the upright position in the event of emergency deceleration during the boost phase and during re-entry.

At high g levels (above 8 g), blackout or dyspnea limit tolerance in positions B and D. Blackout occurs because of the effective positive G associated with the tilt of the trunk in the direction of acceleration. Blackout caused in this way is subjectively indistinguishable from that associated with the usual positive G and, like the latter, can be forestalled with appropriate straining maneuvers. Dyspnea can be partially overcome by the practice of abdominal breathing. Even with this technic, when acceleration exceeds 10 g, the effort required to breathe is so great that it could be maintained by these subjects for only a few seconds (fig. 1).[3]

Of particular interest are the rocket-like acceleration patterns of figure 4. With most liquid fuel rockets, the rate of acceleration increases more slowly at first and more rapidly just before burnout. These patterns result in longer times at low acceleration and shorter times at high acceleration than the experimental patterns with a constant rate of increase of acceleration. The actual rocket acceleration pattern would therefore be expected to be better tolerated by man than an equivalent pattern of the experimental type.

Rather than attempt a reproduction of specific rocket acceleration patterns, as reported in a previous study,[9] the pattern of least tolerance (a constant rate of increasing acceleration) was used in this part of the study. Any actual rocket acceleration pattern which fits within these limits should be tolerated. Further, since the forward-facing subject can tolerate forward acceleration of 4 g for longer than 15 minutes, only those portions of the actual rocket acceleration curves which lie above 4 g need be considered. If these portions of the actual patterns lie within the portions of experimental curves (fig. 4) which are above 4 g, the pattern would be expected to be tolerable regardless of the time required to reach 4 g. Since three peaks of 12 g are tolerable, any combination of three of the individual stages illustrated here would be expected to be tolerated and would achieve a velocity in excess of 18,000 miles per hour.[3]

Re-entry Acceleration Patterns

The magnitude and duration of deceleration associated with re-entry into the earth's atmosphere will depend primarily upon the angle of entry and the aerodynamic characteristics of the vehicle. In general, a large angle of entry and/or a low lift/drag ratio of the vehicle result in a large force acting for short periods of time while a samll angle of entry and/or a high lift/drag ratio result in a small force acting for longer periods of time. Representative g-time curves of each type are illustrated in figure 3.

The deceleration of re-entry can be tolerated in several positions. If adequate restraint is available, the low g (less than 4 g), long-duration patterns are tolerable in any position except head forward (negative G, position F). High g, short-duration forces are best tolerated in position E, B, or D. Early studies indicated a superiority of position D, so great as to justify rotating the pilot 180° to face aft during the re-entry period. In this way, the forces of re-entry would be oriented in the same direction as those of exit, with respect to the pilot and to the seat. More recent studies have indicated that, with adequate restraint, the seated, forward-facing pilot (position E) can tolerate decelerative forces of the same order of magnitude (fig. 1) as the seated aft-facing pilot (position B) or the semisupine aft-facing pilot (position D). This observation was to be expected since tolerance to transverse acceleration is essentially the same in the prone and supine positions.

It would therefore appear that rotation of the pilot to face aft during re-entry might be unnecessary in some patterns. It must be re-emphasized that tolerance of the seated, forward-facing pilot to deceleration is determined largely by the efficacy of the restraint harness. The ideal harness constitutes a support equivalent to a firm outline of the body of the subject.[3]

Prolonged Positive Acceleration

There have been few systematic investigations of the duration of tolerance to positive acceleration. The data summarized in curve C, figure 1 exceeds previously reported limits by a wide margin. These tolerances were demonstrated by employing a rate of onset of acceleration which was slow enough to allow reflex cardiovascular compensatory mechanisms to come into full force before maximum acceleration was reached. A faster rate of onset results in blackout at the usual g levels unless an anti-G suit is used. These observations indicate that velocities in excess of 18,000 miles per hour may be reached by headward acceleration of the seated subject if the magnitude of acceleration does not exceed 5 g. For instance, an acceleration of 3 g, maintained for one hour (figure 1, curve C) would increase velocity by 235,000 miles per hour.[6]

Water Immersion

Preliminary observations suggest that the duration of tolerance of immersed subjects to acceleration up to 12 g is in excess of twice the maximum reported for nonimmersed subjects. Since, as indicated in figure 4, water immersion is not required to tolerate accelerations sufficient to reach orbital velocity, the usefulness of this protective technic is questionable. The tolerance of the immersed subject to acceleration in excess of 12 g has not been investigated.

General Considerations

The rate of increase of acceleration is of some importance in determining both positive and transverse acceleration tolerance.

With transverse acceleration, rates of onset faster than those indicated have resulted in slightly longer (few seconds) tolerances at high g levels. Slower rates of onset have shortened the time of tolerance.[3] The same considerations apply to slower rates of onset with positive acceleration as with transverse acceleration.[6]

In addition to the data summarized in figure 1, it has been reported that 15 g can be tolerated for five seconds in position D if the rate of increase of acceleration is 6 g per second.[4] It appears doubtful that this rate of increase of acceleration will be encountered other than during an emergency. During World War II, two subjects tolerated 17 g for one minute on a German centrifuge. The subjects were in position B. The maximum rate of increase of acceleration possible in this instance was 1 g per 6 seconds. The subjects were reported to have been ill for an indefinite period after the experience. There were no permanent residua.[5] It would appear, therefore, that the tolerance limits indicated in the current studies are well below the level of injury.

A general characteristic of all the curves of figure 1 except F, is the excess tolerance at low g levels, in all positions, over that required to reach orbital velocity. A larger g-time product (greater velocity) can be reached by low g for long periods of time than by high g for short periods. Prolonged low g (below 4) therefore, constitutes one means whereby man can reach velocities in excess of those required for orbit or escape. Arm and leg movements can be effective at these low rates of acceleration.

Although there have been no measurements of performance during the studies summarized here, certain observations are pertinent. A study of the ability of subjects to perform a dual tracking task during three-stage accelerations to peaks of 8, 5.8, and 5.8 g has been reported. The decrement in tracking

ability was so slight that the authors concluded that "select crewmen can be expected to assist in the control of such a vehicle during the critical acceleration phases of the flight."[9] It is generally recognized that highly effective arm movements cannot be executed above 6 g. Wrist and finger movements can be executed up to at least 12 g.

The importance of the post-acceleration symptoms, as observed in these studies, cannot be established. A few of the subjects consistently presented minimal post-acceleration symptoms. Others consistently presented maximal symptoms, and, in still others, the symptoms varied from day to day. In no instance was a subject incapacitated, even immediately after acceleration.[3,8]

The use of the centrifuge to produce acceleration introduces the artifact of angular acceleration of larger magnitude than that expected during exit and re-entry. The effect of this artifact is to increase stimulation of the labyrinth. In none of the accelerations reported here were the runs limited by symptoms of labyrinthine stimulation. It is therefore the opinion of the authors that this artifact did not significantly modify either the magnitude or the duration of the accelerations which were tolerated. After acceleration on the centrifuge, manifestations of labyrinthine stimulation were consistently present (dizziness, vertigo, nausea, and occasionally, nystagmus). It is possible that the post-acceleration symptoms were due, at least partially, to angular acceleration. This premise is supported by the observation of many of the subjects that the symptoms began during the period of deceleration.[3,8] It would seem safe to conclude that the post-acceleration symptoms after linear acceleration would not be more severe than those encountered in these studies.

SUMMARY

Human tolerance limits to the major linear components of the acceleration patterns anticipated in achieving space flight have been reviewed. Acceleration of the anticipated magnitude and duration is best tolerated with the body positioned so that the forces act transversely in a chest-to-back or back-to-chest direction. The optimal body position for exit appears to be a seated position with a 20° inclination of the trunk in the direction of acceleration, with the legs fully flexed (seated, forward-facing). Three-stage accelerations sufficient to reach orbital velocity, with peaks of either 8, 10, or 12 g, are tolerable in this position. With the trunk in the position described in paragraph 3, but with the legs partially extended perpendicular to the direction of acceleration (semi supine position), acceleration peaks to 10 g are tolerated. Such acceleration patterns are also sufficient to reach escape velocity.

Some of the patterns of the deceleration of re-entry can be tolerated in the seated, forward-facing position with the aid of an effective restraint system. Tolerance would be only slightly improved by rotating the subject to face aft during re-entry. Accelerations of less than 4 g are tolerable in either chest-to-back, back-to-chest, or foot-to-head direction for long enough to exceed escape velocity. Prolonged low g acceleration patterns, while not feasible with current propulsion systems, have the potential advantage of enabling man to reach very high velocity (200,000 m. p. h.) and to retain a degree of mobility sufficient to perform limited control functions during the boost phase. Immersion of the entire subject in water enables the attainment of tolerance *times* greater than twice those reached in any other way. The full possibilities of this method of protection have not been explored.

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HEAT STROKE

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THE EMERGENCE of homeothermic animals was a major evolutionary event in that it greatly liberated the animal from its environment. This was accomplished by a thermoregulatory mechanism which maintained a narrow range of body temperature despite wide swings in environmental temperature. In contrast the cold-blooded animal, whose tissue temperature approaches that of the outer world, remained a slave to its environment. The tempo of its activity was largely dictated by the whims of environmental temperature.

Temperature constancy is dependent upon a balance between the heat gain and heat loss of the body. Heat gain is caused by metabolic activity and, in hot climates, radiation, convection, and conduction of heat to the body. Heat loss occurs principally by the evaporation of water from the skin, and surfaces of the respiratory system. In temperate climates considerable heat loss also occurs by radiation and convection of heat from the body. The remarkableness of man's heat-regulating mechanism was dramatically demonstrated in 1775.[1] It was then shown that a man could withstand a temperature of 250°F in an oven for 15 minutes without any ill effects or serious rise in body temperature. Yet a beefsteak was cooked in 13 minutes at the same temperature.

The heat-regulating mechanism can be strained or interfered with during prolonged exposures to heat so that heat casualties will result. The number of victims can reach epidemic proportions, to the point of disrupting military operations. For example, during the summer of 1942, two armored divisions were transported on air-conditioned trains to a desert training site.[2] The daily temperature range of the area was approximately 88°F to 120°F. One division arrived in the latter part of July and immediately set up camp. The incidence of heat casualties was considerable. It was not uncommon that 20 to 30 victims a day were admitted to the hospital. On one day alone, 150 victims were admitted. There were, however, a larger number of casualties who were not admitted but were nevertheless ill enough to be rendered unfit for duty. The division as a whole had become ineffective and could have been an easy prey for the enemy.

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The other division began to arrive in the same area two weeks after the first. The incidence of heat illness was markedly reduced because of the experience gained in the handling of the first.

Incidence

Heat stroke is the most serious of the heat disorders, with reported mortalities of 10 to 80 per cent.[3] Other forms of heat illness—heat syncope, heat exhaustion, heat cramps, and thermogenic anhidrosis—are rarely, if ever, fatal.

In a civilian population most of the heat-stroke victims are in the upper age group, with associated general and cardiovascular arteriosclerosis.[4] In the military most victims are basic trainees, less than 30 years of age, often transferred from their homes in the north to southern training sites.[5] Malamud, Haymaker, and Custer,[6] in a series of 125 fatal cases of heat stroke in the military between 1941 and 1944, noted that one quarter of the victims had been in the Army less than two weeks, and about one half for less than two months.

There have been scattered reports of iatrogenic heat stroke in hospital patients.[7, 8] These were often surgical patients who had been prepared for surgery or endoscopic examination with NPO orders, atropine (which suppresses sweating), and barbiturates (which inhibit the heat-regulating center). In a non-air-conditioned operating room, in a patient wrapped snugly in gowns and the sheets of the operating table, evaporative and convective heat-loss are effectively screened.

Outbreaks of heat stroke are also reported in geriatric and mental institutions.[9] In July 1954, the Midwest suffered a heat wave with temperatures rising above 106°F for 14 consecutive days. On 13 July, with a temperature of 116°F, a Kansas mental institution began having an outbreak of heat illness which included 25 heat-stroke cases. Three of these or 12 per cent died.

Pathophysiology

In some victims, heat stroke seemed to occur precipitously without premonitory symptoms. Yet few heat-stroke casualties appeared during the early part of a heat wave.[3] Apparently there was a subclinical build-up of abnormal physiologic processes within such victims. Existing literature on heat stroke refers to the possibility of a failure of the heat-dissipating center of the hypothalamus. The anterior portion of the hypothalamus contains neurons, susceptible to rising blood temperature and afferent impulses from the peripheral thermoreceptors.[10] The descending pathways of the hypothalamic center pass down the brain stem and set up connections with respiratory and cardiovascular mechanisms of the brain stem and spinal cord. Under conditions of heat exposure, the center evokes increased respiration to the stage of panting and increases peripheral blood flow and perspiration.

It is also possible that an inhibition of metabolic heat production occurs.

Lesions of the anterior hypothalamus such as tumor, infection, hemorrhage, and trauma, can cause inability to adjust to elevated external temperature. It is not uncommon that the body temperature rises to 105°F to 107°F in the terminal stages of cerebral hemorrhage and after surgery for craniopharyngioma.

In view of its important role in body temperature regulation, the hypothalamus has been examined in fatal cases of heat stroke. Malamud, Haymaker, and Custer,[6] however, were unable to demonstrate significant anatomic change in the hypothalamus of 125 fatal cases of military age. This was in contrast to other portions of the brain, especially the cerebellum, which displayed much greater damage in the form of hemorrhage, edema, reduction of neurons, and increase of glial elements. Apparently, the damage within the hypothalamus was on biochemical-biophysical levels, well beyond the range of the microscope.

The biochemical defect may well be in the nature of a central nervous "fatigue." Fatigue refers to the inability of nervous tissue to maintain a maximal excitatory state.[11] The nature of such fatigue is largely unknown. Central nervous system fatigue might result from several causes, such as the using-up of energy-producing materials, depletion of humoral transmitters at synapses, and accumulation of wastes that block nerve transmission.[12] The concept of fatigue can be used as part of a working hypothesis in the pathogenesis of heat stroke. One may picture the hypothalamus under maximal stimulation during exposure to prolonged and severe heat stress. The hypothalamus must constantly evoke the peripheral processes that promote heat loss: maximal sweating, increased peripheral blood flow, and respiration. Should any of the peripheral heat-loss mechanisms be defective, as for example, the congenital absence of sweat glands, or defective sweating resulting from previous brain damage, the hypothalamus is placed under a greater strain than usual and is more readily subject to fatigue.

The hypothalamic fatigue is probably progressive and is reflected peripherally in a gradual diminution of sweating. Many heat-stroke victims note a defective sweating hours or days before the onset of severe neurologic symptoms.[13] Gerking and Robinson[14] demonstrated a 10 to 80 per cent decline in the sweating rate during 6-hour exposures of working men to severely hot environments. The decline was greater in wet than dry heat. Kuno,[15] commenting on these experiments, noted that the sweating rate was rapidly restored with lowering of body temperature under laboratory conditions. He did not believe that this could occur with a peripheral fatigue of the sweat-secreting cells. Rather, the decline was probably caused by a "disorder in the nervous system."

Ladell[16] also found a falling-off of sweat response in an exponential manner with constant skin temperature and rising rectal temperature. After a certain temperature was reached, any further rise in temperature resulted in a decrease in total sweat production. Theoretically, at a rectal temperature of 110°F, the sweating rate is practically nil. Inasmuch as the fall in sweating rate is rapidly reversible, he also did not believe that it was a true fatigue. Ladell concluded that the cessation of sweating in hyperpyrexia is intrinsic in the nature of the sweat-gland response and that there was no need to postulate any pathologic process. He believed that heat stroke is "a purely physiological phenomenon." This is probably an over-simplification. It should be noted that the sweat-gland response has a nervous component. The rapid reversal of sweating rates is not inconsistent with a fatigue of the central nervous system centers. One may consider the fatigue as "physiological" in that it is an intrinsic property of the nervous tissue taking part in the sweating response. This seems to be, however, a purely semantic problem. One can call pneumonia "a purely physiological phenomenon" in that it is intrinsic in the nature of lung tissue to undergo inflammation when invaded by certain organisms. In addition, Ladell's experiments were done with healthy men under controlled conditions and were fairly acute in time, usually a matter of a few hours. Heat-stroke victims, however, are usually exposed to excessive heat loads for several days before the overt manifestation of their illness. Furthermore, a rapid restoration of sweating with a drop in rectal temperature does not always occur in actual heat-stroke victims.

With this background in fatigue, the following sequence of events is postulated:[6] (1)The victim is exposed to an excessive heat load which tends to raise the body temperature. (2) The excessive stimulation tends to fatigue the hypothalamic center. (3) The sweating rate gradually diminishes and the body temperature rises further. (4) Body metabolism is accelerated by the elevated body temperature, thereby producing further heat in the body. (5) Shock ensues as the result of heat damage to the vasomotor centers. The peripheral vasoconstriction accompanying the shock further impairs heat loss. (6) The hyperthermia damages the brain and other organs. Hyperthermia and the anoxia of shock lead to the death of the victim.

There is little mention made in the literature of the mechanism of heat death on the cellular level. Yet this is an important consideration in that the symptomatology and lethality of heat stroke can be traced to the direct effects of heat upon the cell. Most of the work on cellular heat death has been done with unicellular organisms, plants, and cold-blooded animals.[16] The findings may supply clues as to what occurs in human heat-stroke casualties.

Examination of heat-killed cells reveals an increased viscosity of the protoplasm. From this it is assumed that heat kills by

directly causing an irreversible clotting of protoplasm or destruction of the cell enzymes or both. Plants and animal cells, however, can be killed at temperatures which do not cause irreversible protein coagulation or enzyme destruction.[17] Furthermore, there were heat-stroke victims whose temperatures never reached above 103°F to 104°F.[4,16] It seems unlikely that such temperatures can cause irreversible protein coagulation or enzyme destruction. Heilbrunn[19] suggested that heat death is initiated by the action of heat on the cell lipids. The melting point of fats probably has something to do with heat acclimatization. Animals and plants of the tropics have higher melting-point fats than those in northerly climates. The melting point of fats can be raised by subjecting an animal to chronic heat exposure. This has been demonstrated in insects, fish, plants, pigs, and other organisms. Furthermore, the melting point of fats has been correlated with heat resistance—the higher the melting point, the more resistant the animal is to heat stress.[17] Whether the above also applies to humans is not known. If we assume it does, then soldiers could conceivably be made heat resistant and less prone to heat illness prior to entering hot climates. This could be done with diets containing solid fats and carbohydrates which tend to raise the melting point of body fats.

Heilbrunn believed that excess heat tends to dissolve or liquefy fats—especially those with relatively low melting point—situated in the cortex of the cell. As a result, there is a loosening of the chemical bonds between calcium and fat so that an excess of calcium is released into the interior of the cell. The calcium causes severe clotting of protoplasm and death of the cell. Fat solvents, such as ether and alcohol, have the same effect as heat. This, then, suggests that alcohol can augment the effect of an excessive heat load, and thereby predispose one to heat stroke.

The question arises as to why certain individuals in a group exposed to the same heat load are the only ones affected. Could there be some predisposing abnormality in the means of heat dissipation? It is interesting that some heat-stroke victims stated that they "rarely, if ever" perspired.[13] This, then, suggests the possibility of an abnormality in the sweating mechanism. The congenital absence of sweat glands is an obviously predisposing factor, but such cases are rare. One wonders whether there is a relative diminution of sweat glands in those prone to heat stroke. There are racial differences in the number of sweat glands, the Negro having more than the Caucasian. Kuno[15] found a considerable variation among the Japanese. In a study of seven Japanese, the average number of glands per sq cm varied from 143 to 339. Furthermore, there was some correlation between the amount of sweat emitted and the size of eccrine sweat glands. Conceivably, a congenital atrophy of sweat glands in addition to subnormal numbers, could predispose one to heat illness.

There has been, however, no systematic study of the sweat glands in heat-stroke victims.

There are cases on record in which the number of sweat glands was normal, but their functioning was defective.[15] These cases may have had previous brain damage in the form of an encephalitis in childhood. A careful search of the medical history of victims may reveal the occurrence of childhood febrile convulsions, or other abnormal neurologic occurrences, which could account for a functional disorder of the hypothalamic heat-dissipating center.

Chronic degenerative disease may also predispose to heat stroke. Morgan and Vonderahe[20] studied the hypothalamic nuclei in 13 elderly victims and compared them to the hypothalamus of cases not associated with heat stroke. They found a diminished number of cells in the anterior nuclear groups—which are concerned with heat loss—that are apparently the result of previous disease. The posterior groups—concerned with heat conservation—had normal numbers of cells that showed acute pathologic alterations. The normal balance between heat loss and heat conservation probably was tipped in favor of heat conservation in those victims prior to heat stroke. With excessive heat stress, the anterior nuclei were thus unable to evoke sufficient heat dissipation because of previous damage. The pathologic alterations in neurons of the posterior nucleus suggested intense over-activity. Possibly there was a lessening of normal checks and inhibitions to the extent that heat production and conservation were accelerated rather than inhibited. The findings of Morgan and Vonderahe are at variance with Malamud, Haymaker, and Custer[6] who found no significant anatomic changes in the hypothalamus. The probable reason is that the latter studied specimens from young men of military age, and the former used material from victims over 50 years of age, who had had chronic degenerative disease (chronic vascular sclerosis, syphilis, or chronic alcoholism).

The degree of hydration may be an important variable in predisposition to heat stroke. Lichton,[21] working with subjects acclimatized to heat for 2 weeks, demonstrated that there was a rise in the threshold for sweating and also a fall in the rate of sweating in acute water deprivation. The rectal temperature in the dehydrated subjects reached high levels at much lower effective air temperatures than was the case in normally hydrated subjects. Dehydration is said to set the hypothalamic thermostat at a higher than normal level,[22] therefore adding to the vicious circle of hyperpyrexia.

Obesity makes an individual more prone to heat stroke.[23] Death because of hyperthermia is three-and-one-half times more likely among those who are 40 lbs overweight than among individuals who are overweight by only 10 lbs.[23] Inasmuch as metabolic heat

is produced proportionately to bulk of tissue and is lost proportionately to surface area, a low volume-to-area ratio is a great liability in a hot environment.

In a series of 74 patients, 23 or 30 per cent gave a history of alcoholic intake prior to the onset of heat stroke.[4] Five subsequently died. Alcohol stimulates the sweating mechanism through its central action.[15] Conceivably, such an added stimulus to an already overburdened hypothalamus may in part be responsible for its failure.[3] However, the work of Heilbrunn, discussed above, suggested that alcohol augments the effect of heat by its action on cell lipids.

There are then several abnormalities that may predispose one to heat stroke: abnormalities in number and size of sweat glands; previous hypothalamic damage resulting from encephalitis, febrile convulsions, chronic degenerative disease, et cetera; acute water deprivation; obesity; acute alcoholism, and abnormal fat metabolism.

The absence of abnormal anatomic or physiologic predispositions make the occurrence of heat stroke less likely. Heavy physical work, improper clothes, high humidity, excessive radiant-heat loads, et cetera, can also, in and of themselves, lead to heat stroke.

Clinical Features

In Austin and Berry's series of 100 cases,[4] only 7 per cent stated that their symptoms began suddenly; 28 per cent had symptoms for less than 24 hours. The majority had premonitory symptoms lasting 2 to 5 days. In a series of 125 fatal cases,[6] 71 per cent were acute without warning, 21 per cent had prodromata of a few minutes to hours, and 8 per cent had symptoms lasting several days prior to heat stroke.

Prodromata in the order of their frequency[6] were faintness, staggering gait, dizziness, headache, nausea and vomiting, purposeless movements, muscle cramps, choking, difficulty in swallowing or speaking, numbness of extremities, drowsiness, restlessness, mental confusion, dryness of the mouth, excessive thirst, anorexia, and diarrhea. Three to four per cent gave a history of previous heat illness.[4,6]

Death occurred within 24 hours in approximately 70 per cent of 125 fatal cases, the remaining surviving as long as 12 days.[6] Of the 17 deaths in Austin and Berry's series,[4] 7 occurred within 24 hours, 7 within 1 to 6 days, and 3 within 6 to 13 days. The cause of death was acute, left ventricular failure in 8 of 17 fatalities, and uremia in 2. The remaining 7 deaths resulted directly from the effects of heat.[4]

The temperature of a heat-stroke victim does not necessarily rise above 106°F. Such cases have been termed "borderline

hyperpyrexia" by Ladell, Waterlow, and Hudson.[18] Of 100 cases, 39 were never observed with temperatures over 106°F;[4] 10 per cent of these died, in contrast to a fatality of 21 per cent in victims with temperatures above 106°F.

Patients seen initially with subnormal or slightly elevated temperatures usually showed a subsequent rise to levels exceeding 106°F. After the hyperthermia is reduced by some cooling measure, there is often a secondary rise or several rises in temperature—probably an indication of the instability of the hypothalamic center.

In a study by Malamud, Haymaker, and Custer,[6] blood pressure was normal in 35 per cent of 55 fatal cases. The remainder had systolic pressures ranging from 40 - 100 mm Hg and diastolic 0 - 60 mm Hg. The presence or absence of shock levels was a better prognostic index than the degree of hyperthermia. In cases in which survival was longest, shock was either successfully controlled at the very onset or was not appreciable until the terminal stages. Approximately one third of those with systolic pressures below 100 mm Hg will die.[4]

The work of Daily and Harrison[24] on experimental heat stroke in animals suggested that initially an excessive heat load caused a cutaneous vasodilation with compensatory splanchnic vasoconstriction. The shock in heat stroke occurs when the vasomotor centers, incapacitated because of thermal injury, fail to affect the compensation. In addition, the splanchnic vessels may dilate as a result of the direct stimulus of heat or acid metabolites, or both.

Complete or marked suppression of sweating is one of the cardinal manifestations of heat stroke. This finding is important in differentiating heat stroke from other diseases. Occasional victims, however, show hyperhidrosis.[6,25] This may be because of the co-existence of heat stroke with heat exhaustion in which heavy perspiration is the rule.

Disturbances in consciousness from lethargy to coma are usual. Only 2 per cent exhibited normal levels of consciousness.[4] There may be generalized or Jacksonian-type convulsions, muscle rigidity or flaccidity, meningeal signs, pyramidal signs, opisthotonos and decerebrate rigidity.[6] The higher the temperature, the more severe the central-nervous-system signs.

Neurologic sequelae, particularly of the cerebellar type, are not uncommon in heat-stroke survivors.[26] In 3 of 20 survivors, or 15 per cent, cerebellar symptoms persisted.[16] There may be ataxia, nystagmus, in-co-ordination of the extremities, athetoid movements, speech disturbances, irritability, headaches, psychosis, apathy, disorientation, hemiplegia, et cetera.

Other possible accompaniments of heat stroke are jaundice, pneumonia, muscle cramps and tetany, uremia, anuria, and purpura.[6]

Laboratory Findings

In a study of 15 cases,[27] an initial hemoglobin of 11.6 to 16 grams per 100 ml (average 13.6) was found. This decreased in one week to 8.2 to 13.8 (average 10.7). There was no obvious blood loss. An increased rate of red blood cell destruction was detected in 10 patients. According to Ladell, Waterlow, and Hudson,[16] hemodilution is usual in the later stages of heat stroke because water accumulates in the body upon cessation of sweating. White blood cell counts were normal or elevated.[6]

Thrombocytopenia was present in all cases.[6] In 6 fatal cases there was a progressive fall in platelets, but in one there was a return to normal values before death. Prothrombin time may be normal or prolonged, probably as a result of liver damage.[6] Prolonged clotting time was found in 5 of 15 cases.[27] The impaired coagulation of blood accounts in part for the widespread hemorrhage found at autopsy.

Wright, Reppert, and Cutting[28] demonstrated capillary fragility by means of the tourniquet test. They suggested that the capillary dilation that occurs on exposure to heat may reach a pathologic state permitting red blood cells and plasma to extravasate. They postulated that in addition to a decrease in prothrombin time or platelet count, or both, capillary damage contributes to the purpuric manifestations of heat stroke.

Blood-urea-nitrogen determinations were performed in 61 patients;[4] 29 showed blood urea nitrogen above 20 mg per cent; 9 deaths occurred in this group, indicating the seriousness of this finding. Elevated blood urea nitrogen may not necessarily reflect kidney damage, but rather generalized tissue destruction.

In heat stroke, blood-electrolyte levels were usually not remarkable unless kidney function was impaired.[4] Potassium tended to be decreased, but Austin and Berry[4] found 4 of 44 cases with hyperkalemia, all of whom died. The hyperkalemia may have been caused by intravascular hemolysis of red blood cells and widespread tissue destruction. Blood chlorides were very variable, with a range of 90 to 138 mEq/l in 47 determinations. Sodium and CO_2 combining power also showed wide variations; the former, 124 to 171 mEq/l in 43 patients; the latter, 11 to 34 mEq/l in 38 determinations.

Liver-function tests may show parenchymal damage.[6] The cerebral spinal fluid may occasionally show elevated pressure and protein values.[6] The urine not infrequently showed albuminuria, red blood cells, white blood cells and hyaline-granular casts. Electroencephalographic and electrocardiographic studies revealed nonspecific brain and myocardial damage which are usually temporary in survivors.[6]

Pathology of Heat Stroke

Heat stroke affects both the central nervous system and the viscera.[6] The central nervous system showed a progressive destruction of neurons and a proliferation of glial elements. These changes were most marked in the cerebellum, cerebral cortex, and basal ganglia, but not in the hypothalamus or the rest of the brain stem. Malamud, Haymaker, and Custer[6] believed that the cellular changes were the direct effect of hyperthermia. They noted that the degree of cellular pathology corresponded with the duration of survival after the onset of hyperthermia, not to the length of illness in those cases where elevated temperatures were a terminal event. There was also edema, congestion and petechial hemorrhage, most commonly about the third ventricle, aqueduct and fourth ventricle. Malamud, Haymaker, and Custer[6] were of the opinion that those changes were secondary to shock. They observed a direct relationship between the severity of shock and the degree of hemorrhage. Furthermore, the distributional pattern of hemorrhage was analogous to that occurring in diseases associated with shock, but not hyperthermia. The coagulation defects of heat stroke also contributed to the hemorrhage.

The abdominal and thoracic viscera showed both hemorrhage and parenchymal damage.[6] The longer the survival, the more widely disseminated the hemorrhage. No correlation could be made between blood pressure levels and the degree of hemorrhage. Coagulation defects undoubtedly contributed to the hemorrhage. The structures most commonly involved were the lungs, epicardium and endocardium, peritoneum, gastrointestinal tract, pleura, skin, and heart muscle.

The longer the survival, the more severe the parenchymal lesions. Structures involved in order of frequency[6] were megakaryocytes, heart muscle, lungs, kidney, liver, and adrenal cortex. The involvements accounted for the clotting defects, EKG changes, lobular pneumonia, lower nephron nephrosis, and jaundice, sometimes accompanying heat stroke.

TREATMENT

It is universally agreed that reduction of body temperature as rapidly and safely as possible is needed to reverse the manifestations of heat stroke. Evaporative cooling has been recommended as the best means.[24] After the patient had been wrapped in a wet sheet, several electric fans were directed at him while his skin was massaged through the wet sheets. The purpose of the massage was to aid the circulation through the constricted peripheral vessels. This therapy was said to be preferable to the ice bath because the evaporation of one gram of water removed seven times as much heat as the melting of one gram of ice. Also, the ice bath was said to cause cutaneous vasoconstriction, thereby interfering with heat loss.

Despite these objections, the ice bath has been found to be more effective than evaporative cooling. Daily and Harrison[24] compared evaporative cooling and ice-bath treatment on experimentally induced heat stroke in mice. They found more survivors in the ice-bath treated group. In 25 cases of heat stroke, Ferris and others[3] were able to effect a temperature reduction to 102°F in 9 to 40 minutes by means of the ice bath.

After immersion in ice water, a dramatic change in clinical appearance may occur with falling temperature. Abnormal blood pressure may approach normal levels without intravenous therapy. Patients who were unconscious and flaccid may become rigid and delirious. Considerable effort may be required to prevent the patient from escaping the tub or injuring himself. Some may require restraint for several hours after cooling.[3]

It is advisable to cool the patient to no lower than 101°F to 102°F. In one patient treated by ice bath, body temperature dropped to 99°F before removal from the ice water. His temperature subsequently fell to 96°F and the patient died in shock.[3]

Shock must also be treated. The vascular collapse is primarily peripheral, but cardiac failure may be present in elderly patients. One can treat these by the usual methods of intravenous fluids, Levophed, and digitalis. Levophed should be avoided during hyperthermic periods, as it interferes with heat loss by inducing peripheral vasoconstriction. Oxygen therapy is probably valuable when peripheral failure, pulmonary edema, or cyanosis are present. Wright, Reppert, and Cutting[28] recommended vitamin K and early blood transfusions in view of the tendency to hemorrhage. Serious blood loss, however, is rarely a problem in heat stroke. It is not uncommon that bronchopneumonia complicates heat stroke.[6] Prophylactic antibiotics are therefore in order.

After the emergency state has passed, the patient's temperature must be watched because of possible rebound to hyperthermic levels.[4]

The efficacy of adrenal cortical hormones in control of hyperpyrexia has not been definitely demonstrated. Adrenal hormones do have an antipyretic effect and have been used therapeutically to reduce the pyrexia of various infectious diseases.[29] It has been suggested that adrenal cortical hormones act centrally through the hypothalamus.[30] Furthermore, adrenal cortical damage can occur in heat stroke.[6] Ingraham, Matson, and McLaurin[31] were able to prevent postoperative hyperthermia in 6 patients receiving cortisone after surgery for craniopharyngioma. Hypothalamic damage often occurs during this type of surgery. Waugh[29] attempted to determine if cortisone would show an antipyretic effect in heat stroke. He found that orally administered cortisone in one case and parenteral cortisone in another were without effect on either the hyperpyrexia or anhidrosis of heat stroke. Austin

and Berry[4] gave cortisone to 6 severe cases, but were unable to come to any definite conclusions.

SUMMARY

1. Heat casualties can significantly reduce the strength of a military unit.

2. Heat stroke occurs largely in basic trainees transferred from their homes in northern states to southern training camps.

3. An acquired or congenital impairment of heat loss may predispose one to heat stroke. Some of these possibilities are discussed.

4. Heat illness results from a disparity between heat gain and heat loss of the body.

5. Heat stroke is initiated by an excessive heat load which fatigues the hypothalamic heat-dissipating center. Impairment of heat loss and a serious rise in body temperature follow.

6. The excessive body heat probably damages tissue cells by its action on cell lipids.

7. The main clinical findings are hyperthermia, absence of sweat, neurologic signs, and shock.

8. Heat stroke affects both the central nervous system and the abdominal and thoracic viscera. The cerebellum displays the greatest damage in the central nervous system.

9. The degree and duration of hyperthermia and shock are the principal prognostic signs of heat stroke.

10. Laboratory findings are generally non-specific. Low platelet counts occur early because of heat damage of megakaryocytes.

11. Treatment consists principally of temperature reductions and control of shock. The efficacy of adrenal hormones has not been demonstrated.

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EXTRACORPOREAL CIRCULATION FOR OPEN HEART SURGERY IN A NAVAL HOSPITAL

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THE institution of a program for open heart surgery has engendered teamwork and a great deal of effort on the part of many members of the group. The program started three years before it was employed in the first clinical case here, with the experimental use of various types of pumps and oxygenators. This phase of the work was carried out in several animal laboratories in civilian institutions until the past year, when our own surgical research laboratory was commissioned. We believed that we should not use the technic of extracorporeal circulation on a patient until we could repeatedly use it successfully in dogs.

A cardiopulmonary function laboratory, with the availability of cardiac catheterization, is a prerequisite. The institution of such a laboratory is the first step in this program, and we were fortunate inasmuch as such a laboratory has been functioning in this hospital for the past 10 years.

The oxygenator we are currently using (fig. 1) is of the rotating disk type developed by Dennis and associates.[1] It consists of from four to ten 18- by 18-mesh stainless steel screen disks that are 47.5 cm in diameter. These disks are rotated at 24 r. p. m. within a steel tank to which 4 to 10 liters of oxygen per minute are added.

The oxygen is heated to 39° to 40°C to minimize heat loss in the machine. Each disk will completely saturate 400 ml of blood

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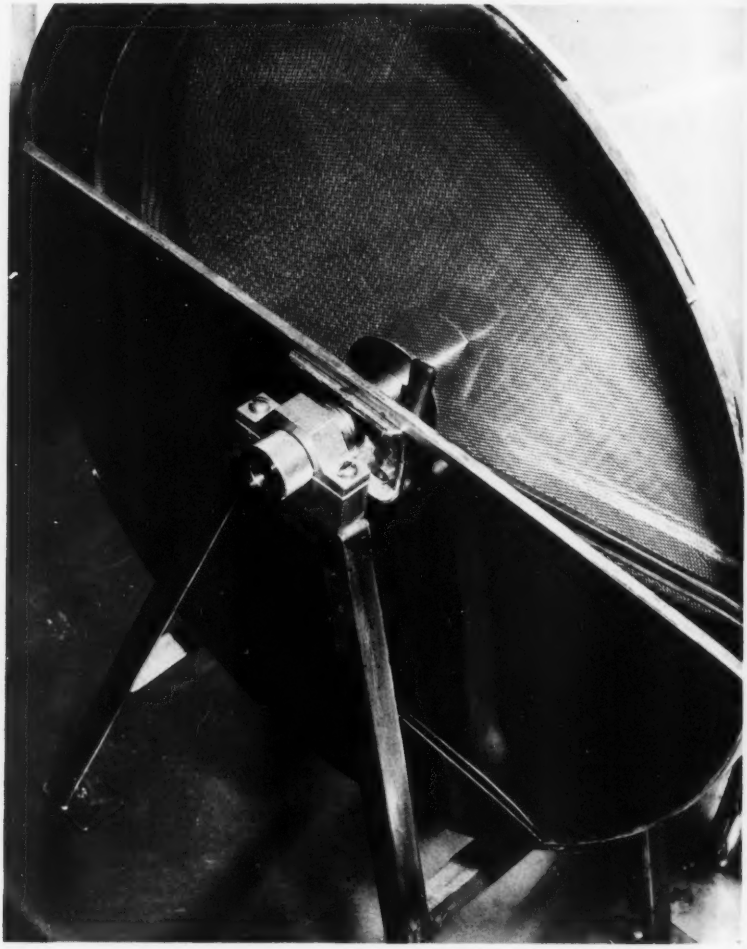

Figure 1. Oxygenator. The rubber tip seen at the center of the disk distributes the blood on each screen. The lower section of the distributor tube delivers the oxygen.

per minute, giving a total capacity of 4,000 ml per minute when 10 screens are used.

The blood drains by gravity from each vena cava directly through a distributor to the center of each disk. The blood films out over the rotating disks and the oxygen and carbon dioxide exchange takes place. The saturated blood then drops to the bottom of the tank. The inside of the tank is lightly coated with a silicone antifoam. From the tank, the blood passes into the reservoir and then into the pump.

All of the connecting tubing and the reservoir are made of polyvinyl plastic, and the pump tubing is made of gum rubber. The connectors, which are made of very carefully polished stainless steel, are designed to give minimal turbulence. No filter is used with this system. The materials used for the construction of the machine were so selected that it could be sterilized by autoclaving.

The pump (fig. 2) that is utilized in our apparatus is of the multiple finger cam-actuated type.* This is a completely occlusive pump which is simple and reliable. With this type of pump, the flow rate is calibrated before the perfusion is started and then does not change with changes in the patient's blood pressure.

For the anesthesiologist, there are essentially two problems that are created during perfusion. First, there is a loss of anesthetic control because the anesthetic agents are diluted by the blood in the pump-oxygenator. Secondly, cerebral hypoxia may develop during perfusion. It therefore is important to guard against drug depression, which will aggravate the hypoxic cerebral depression. Because of the above-mentioned problems, anesthesia should be light. The relaxant drugs can be used to control the patient's movements without excessive use of depressant drugs.

When we were satisfied with the technic of perfusion and were confident that we, as a team, could use it successfully, the procedure was carried out in our first clinical case on 21 May 1958.

CASE REPORT

A 6-year-old girl gave a history of frequent colds, a tendency to tire easily, and difficulty in keeping up with her playmates. No history of squatting was elicited, nor were there any episodes of cyanosis, syncope, or convulsions.

Physical examination revealed a girl who was small for her age. Blood pressure in the right arm was 112/70 mm Hg; that in the left arm, 114/70; and in the left thigh, 120/80 mm Hg. Abnormal physical findings were limited to the cardiac examination. The cardiac apex was 1.5 cm lateral to the mid-clavicular line in the fifth left intercostal space. A prominent systolic thrill was felt over the entire precordial area, and was most marked at the left sternal border in the third intercostal space. A harsh grade 3 systolic murmur was heard over the entire precordium, but was loudest in the second left intercostal space at the sternal border. The second pulmonic sound was louder than the second aortic sound, and both were clearly defined. The rhythm was regular. Radial and femoral pulses were readily palpable.

*Sigmamotor, Inc., Middleport, N. Y.

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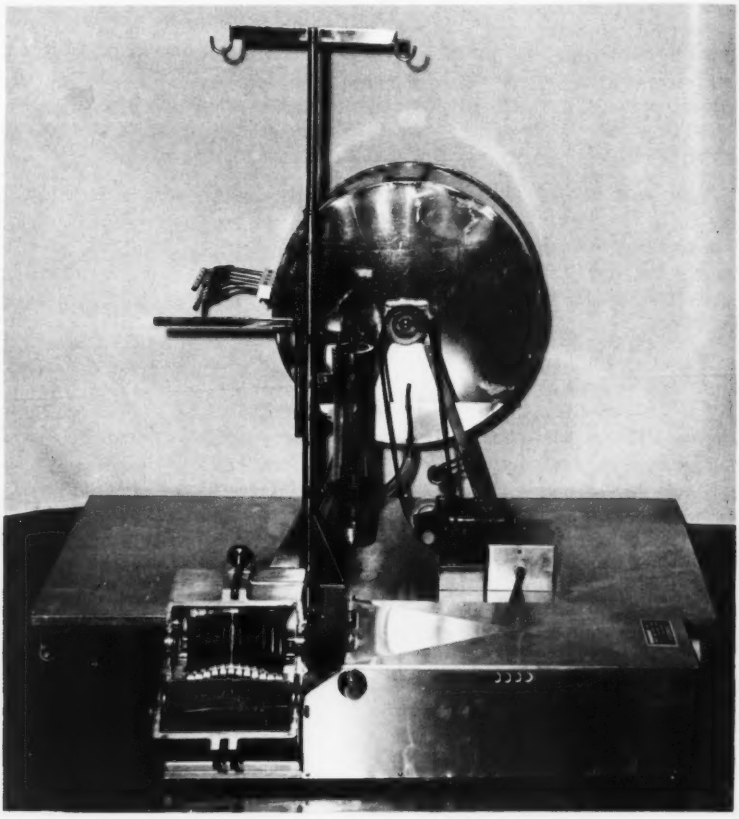

Figure 2. Pump and oxygenator. The TM-2 Sigmamotor finger pump is in the foreground. The oxygenator is the four-screen model with 1,600-ml per minute capacity. The plastic reservoir also serves as a bubble trap.

The electrocardiogram showed evidence of right ventricular hypertrophy, clockwise rotation, and incomplete right bundle branch block. A diastolic overload pattern also was present. Roentgenograms of the chest showed cardiac enlargement and plethoric lung fields. Angiocardiography further defined the cardiomegaly by showing an enlarged right atrium and ventricle, with a normal-sized left atrium and ventricle. Late reopacification of the pulmonary artery occurred, indicating a left to right shunt. The mean pressure in the right atrium was 10 mm Hg. Pressure in the right ventricle was 43/6 and in the pulmonary artery 30/9. The mean wedge pulmonary artery pressure was 7 mm Hg. Oxygen saturation studies indicated the presence of a large left to right shunt at the atrial level.

The preoperative laboratory studies revealed essentially normal blood counts and urinalysis. Bleeding time, clotting time, prothrombin time, platelet count, erythrocyte fragility studies, and blood fibrinogen level were normal.

At 0630 on the morning of the operation, 22 donors from Fort Totten reported to the blood bank. These soldiers had previously been examined and screened and their blood typed and cross-matched with the recipient's blood. Some of the blood drawn was collected in standard A-C-D solution and the remainder in siliconized bottles that contained 20 mg of heparin in 25 ml of saline solution to 475 ml of blood. This blood was immediately sent to the operating room, where it was placed in a water bath at 37°C.

The child was anesthetized with open drop fluothane (brand of trifluro-chlorobromoethane) followed by endotracheal intubation, which was aided by Cyclaine Hydrochloride (brand of hexylcaine hydrochloride) spray of the larynx. With the patient asleep, a polyethylene tube was placed in a saphenous vein for clysis and for the recording of venous pressure. Both femoral arteries were exposed. One received a plastic catheter for the continuous recording of direct arterial pressures. The other received the arterial cannula from the pump. The latter catheter was not inserted until the patient had been heparinized.

The chest was opened with a transverse anterior incision, going through the third interspace on the left side and the fourth interspace on the right with transverse division of the sternum. The thymus gland was dissected from the pericardium, which then was opened widely with a T-shaped incision. Twill tapes were placed about each vena cava intrapericardially in preparation for inflow occlusion. The atrium was explored through the right auricular appendage, and a large secundum type of defect was revealed. The mitral valve, which could be easily felt through the defect, was essentially normal, as was the tricuspid valve. The pulmonary venous drainage was all into the left atrium.

After 27 mg of heparin (1.5 mg per kilo) was injected into the atrium, two No. 22 Bardex catheters were inserted in the atrium and secured with purse-string sutures. These catheters were manipulated into each vena cava, and when the cannulae were secured by the tapes, the pump oxygenator was started. As the machine functioned well from the start, the atrium was promptly opened, with an incision extending from one vena cava to the other without actually opening the vein walls.

The defect, which measured about 5 by 2 cm and was immediately adjacent to the coronary sinus and the Thebesian veins, was closed with a combination of continuous and interrupted silk sutures, size 00 and 000. When the defect was closed and it was ascertained that the coronary sinus had not been compromised, the atrial wall was closed with a single, continuous suture. The tapes about the cannulae were released and the perfusion stopped after 23 minutes. When the

cannulae were removed, 54 mg of protamine sulfate (3 mg per kilo) was administered slowly.

Blood samples were obtained prior to the actual perfusion, at 10-minute intervals during the perfusion, and at its termination. These were examined for plasma hemoglobin, whole blood hemoglobin, cell morphology, hematocrit, platelet count, red cell fragility, fibrinogen concentration, protamine activity, clot retraction, pH, and oxygen saturation. These results are tabulated in table 1.

A remarkable blood homeostasis was maintained during the perfusion; the drop in the platelet count did not interfere with an excellent clot retraction, which was normal after giving the patient the protamine. Protamine titrations by a rough qualitative method gave an excellent end point for blood coagulability of seven minutes after the protamine was administered. Morphologic examination of the cellular elements of the blood samples revealed minimal changes. In general, no major changes were noted in the blood during or after perfusion.

For anesthesia, the patient was maintained with Pentothal Sodium (brand of thiopental sodium), Demerol Hydrochloride (brand of meperidine hydrochloride), and nitrous oxide. Circle absorber, semiclosed technic was used to administer the nitrous oxide. The Pentothal and Demerol were given intravenously in divided doses. A total of 175 mg of Pentothal and 30 mg of Demerol were used. All agents were stopped as soon as perfusion started.

During perfusion, the lungs were kept inflated with a mixture of helium and oxygen. Controlled respiration was carried out throughout the entire procedure, including the period of perfusion.

Electroencephalographic tracings were made throughout the procedure, and at no time was the tracing flat. This indicated that circulation to the brain was adequate.

Arterial pressure, as recorded by the direct method, never was less than 100 mm Hg and during most of the perfusion was 120 mm systolic.

At the completion of the procedure, the heart demonstrated a 2:1 block with a ventricular rate of 40. A silver-coated, stainless steel wire was sutured into the left ventricular wall, then threaded through a polyethylene catheter and brought out through the chest wall. A similar wire was sutured into the anterior chest wall. These wires were inserted so that they might be connected to an external pacemaker if cardiac arrest occurred or if the ventricular rate became too slow. The chest wall was closed after two No. 40 catheters had been placed in each pleural cavity and connected to underwater suction.

Upon completion of the operation, the patient's temperature was 90°F, and the pulse rate was 46 per min. There was a complete heart block with atrioventricular dissociation and idioventricular rhythm. The temperature rose gradually to normal with the aid of an electrically heated blanket, and as the temperature rose the blood pressure and ventricular rate also increased. The patient was taken to the recovery

TABLE 1. *Results of laboratory tests made on samples of blood drawn at various times during the operative procedure.*

Specimen number	Time taken	Remarks	Plasma hemoglobin (grams/100 ml)	Blood hemoglobin (grams/100 ml)	Arterial pH	Hematocrit (ml/100ml)	Platelets (per μl)	Erythrocyte fragility hemolysis		Fibrinogen	Protamine		Oxygen saturation (per cent)
								Begins** (% Na cl)	Complete*** (% Na cl)		Activity (gamma/ml)	Clotting time (min.)	
1	1036	Blood already agitated for 20 minutes in machine	0.132	12.7	7.75	42	484,000	0.04	0.26	4 plus	40	7.5	99.1
2	1206		0.184	12.9		42	259,000	0.40	0.28	4 plus			
3	1236	Perfusion started	0.160	13.2		42	189,000	0.40	0.28	4 plus			
4	1241		0.136	13.0	7.71	42	264,000	0.44	0.28	3 plus			97.7
5	1246		0.150	13.0		42	304,000	0.44	0.28	2 plus			
6	1251												
7	1258		0.150	13.0		42	257,000						
8	1304	Perfusion completed	0.160	12.7	7.72	42	247,000			2 plus	50	6.5	100
9*	1333	Blood drawn 15 minutes after administration of 54 mg of protamine sulfate									10	7	

*This blood clotted spontaneously 8 minutes after having been drawn.
**Control 0.44%
***Control 0.28%

room with a blood pressure of 106/60 mm Hg and a ventricular rate of 72 per min. The electrocardiogram then showed nodal rhythm, and the patient was breathing well, unassisted.

For the first 5 days, a medical officer was in continuous attendance at the bedside. The electrocardiogram was constantly monitored with a cardioscope, and a pacemaker, to be used in the case of cardiac arrest or marked bradycardia, was in the room.

During the night following the operation, the patient's ventricular rate increased to 110-120 per min., and the blood pressure was maintained at levels of 110/70 to 110/80 mm Hg. She was breathing well and was mentally alert. There were no neurologic deficits. The urinary output was good and the urine was clear. The hematocrit was 38 ml per 100 ml and the hemoglobin was 13.2 grams per 100 ml at 2300 following the operation. A roentgenogram of the chest showed the lungs to be expanded, with no abnormal accumulation of fluid in the pleural cavities. There was no oozing from the wounds. The blood pH was normal and the plasma hemoglobin was essentially normal.

On the first postoperative day, the two lower thoracotomy tubes were removed. The patient's vital signs were within normal limits, but it was apparent that she was not effectively raising her bronchial secretions. There were coarse rales bilaterally and the respiratory rate had increased. She was placed in a croupette. Attempts at tracheal aspiration were not effective. Roentgenograms of the chest revealed a right upper lobe atelectasis with some questionable pneumonitis.

On the second postoperative day, direct laryngoscopy and tracheal aspiration were carried out, and a considerable amount of mucopurulent secretion was removed. Roentgenograms following the tracheal aspirations showed little improvement in the atelectasis and pneumonitis. Later during the day, the temperature rose to 102°F and the respiratory rate increased to 40-50 per min. A tracheostomy was performed and a large amount of secretion was removed.

On the third postoperative day, the patient's condition had greatly improved. Her respirations had become less labored and the breath sounds had assumed an almost normal character. Roentgenograms of the chest showed marked improvement of the atelectasis and pneumonitis. The cardiac rate was 115-130 per min. and there was still a nodal rhythm.

By the fourth postoperative day, the patient's condition was considered good. Respirations were normal and she was taking fluids orally in adequate amounts. She continued to improve, and the following day was out of bed and an electrocardiogram showed a normal sinus rhythm. On the ninth postoperative day, the tracheostomy tube was removed. The lungs were clear and the temperature was normal. All of her wounds were healing well and she was ambulatory. The thrill and loud systolic murmur that were present preoperatively were no longer present. There was obvious decrease in the heart size, with a regular

sinus rhythm. Roentgenograms showed the lungs to be essentially normal.

By the twelfth postoperative day, all the sutures had been removed and the wounds had healed per primum. The ventricular wire was removed from the heart with no difficulty. The patient was ambulatory, asymptomatic, and considered to be well. She was discharged to her home on the fifteenth postoperative day.

SUMMARY

The institution of an extracorporeal program for open heart surgery requires co-ordinated teamwork and a tremendous effort on the part of each member of the team. It also requires intensive laboratory work, inasmuch as the laboratory is called upon to perform time-consuming and difficult procedures. Only after repeated success in the animal laboratory with the entire team employed, and after sufficient confidence has been obtained in the use of the heart-lung bypass procedure, can one feel justified in the use of this procedure in clinical cases.

A successful clinical application of extracorporeal circulation and open cardiotomy for the repair of an atrial septal defect is reported.

ACKNOWLEDGMENT: The authors wish to thank Dr. Clarence Dennis and Dr. J. Maxwell Chamberlain for their encouragement and assistance, and the many members of the staff of this hospital who gave selflessly of their time and effort.

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EXCHANGE TRANSFUSION FOR ACUTE POISONING IN CHILDREN

OGDEN C. BRUTON, *Colonel, MC, USA*

EXCHANGE transfusion in the newborn period to prevent or remove dangerous amounts of circulating indirect bilirubin is a common procedure in pediatric practice. The indications for and the technic of this procedure is well learned in almost every pediatric residency.[1] Dangerous amounts of other circulating substances are encountered with disturbing frequency in older children. Occasionally the amount ingested and the substance cannot immediately be determined; however, the necessity for additional treatment may be recognized. Because dialysis of almost any nature is technically difficult and not available to most hospitals, exchange transfusion has been found to be effective, without undue hazards, and may be quickly provided in selected cases of poisoning.[2-4] As a reminder of this useful tool as a possible lifesaving measure, the following three illustrative cases are presented.

CASE REPORTS

Case 1. This two-year-old white boy was apparently well until 1100 hours on the day of admission, when the mother noted he was holding a bottle of Nembutal (brand of pentobarbital sodium) capsules, with an estimated 17 to 20 100-mg capsules missing from the bottle. When the capsules could not be located and the patient became very lethargic, he was rushed to the hospital, where at 1130 hours he was comatose, with markedly depressed respirations and absent lid and corneal reflexes. Immediate stomach lavage was instituted and was continued until 1300 hours.

Because of the patient's failure to respond to painful stimuli, marked depression of respirations, and the probability of having ingested a large amount of the drug, it was decided an exchange transfusion should be done. By 1345 hours the transfusion had been started through a polyethylene catheter placed in the right femoral vein. The exchange was stopped at 1620 hours, after 2,290 ml of whole blood had been exchanged. Midway of the procedure the patient responded to painful stimuli, later moved his extremities, and toward completion yawned several times. His vital signs re-

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mained stable and were reported as pulse rate, 100 per minute, respirations, 14 per minute, temperature, 97.2°F, rectally, and blood pressure, 116/62 mm Hg., at completion of the procedure. Infrequent pharyngeal suction was necessary for 2 to 3 hours and respirations gradually increased to 34 per minute. At 1935 hours he was reported to cry and resist restraints. The night was spent in fairly quiet sleep and he seemed normal the following day. On prophylactic penicillin and streptomycin the wound healed rapidly and there were no further complications.

Case 2. This three-year-old white girl was apparently well until she came in from play at approximately 1100 hours on the day of admission to the hospital, and complained of abdominal pain, and promptly vomited orange-colored fluid several times. Upon arrival at the hospital her lower legs and feet were stained a brownish-orange by vomitus, but otherwise there were no abnormal findings. Stomach lavage was started immediately and continued for 45 minutes when the return had almost cleared. It was noted at 1230 hours that her lips appeared slightly cyanotic; however, this was at first thought due to the child's crying and resisting lavage. The mother had been sent home to seek out the possible ingesta. She returned with an empty Pyridium (brand of phenylazo-diamino-pyridine hydrochloride) bottle which was found beside a garbage can in the neighbor's yard. The neighbor admitted discarding a bottle of Pyridium, containing all of the prescribed amount for a kidney infection, which she had not used.

On completion of lavage the patient was noted to be quite lethargic. Lethargy and cyanosis became progressively more marked without improvement with oxygen therapy. Venous blood appeared extremely dark and failed to change color on shaking in test tube or on standing. Methemoglobinemia was an obvious diagnosis. A catheterized urine specimen had the appearance of a concentrated solution of iodine. The child's appearance and reaction seemed to warrant immediate further treatment to prevent possible serious sequelae. Intravenous methylene blue was not available. Exchange transfusion was recommended and was started at 1445 hours. After 1,400 ml had been exchanged, the patient's skin, lips, and nail beds gradually became a normal pink color. She tolerated exchange of 2,000 ml blood well, and on prophylactic penicillin and streptomycin was asymptomatic the following day. Chemical analysis for methemoglobin on pre-exchange blood done two days post-exchange revealed methemoglobin 40 grams per 100 ml.

Case 3. This 28-month-old girl was brought to the hospital with a history of ingestion of a minimum of 48, 1¼-grain aspirin tablets and an undetermined number of 5-grain tablets approximately one half hour previously. She was immediately given gastric lavage following which the mother thought the child was rather depressed. On admission there was no other abnormal observation made. A blood salicylate level was reported as 73 mg per cent two and one-half hours after

ingestion, and at four and six hours after ingestion as 121 mg per cent and 160 mg per cent respectively. The blood CO_2 was reported 10 mEq and the attending physician believed there had been progressive signs of intoxication although her temperature remained normal. Appropriate fluids were started intravenously and the patient had voided five times; however, she also vomited. Eight hours after the ingestion, exchange transfusion was decided upon and was completed by 12 hours after ingestion with 1,000 ml whole blood being exchanged. The blood salicylate level was reported as 24.3 mg per cent immediately post-exchange. The procedure was well tolerated and her convalescence was uneventful.

DISCUSSION

During 1956 and 1957 acute poisoning in children was responsible for 142 admissions to Tripler U. S. Army Hospital, or 2.6 per cent of the total children admitted for all causes. This increased incidence over the usual expected rate of approximately 1 per cent[5] probably is explained by the increased exposure of children, living in temporary circumstances, to poisons of various kinds, and in weather permitting year-round outdoor play. There were no deaths as a result of poisoning during the period of study, and exchange transfusion was not considered necessary in other children. However, subsequent to the period of study there has been one death, caused by ferrous sulfate poisoning in which exchange transfusion was considered indicated, but the child expired before the treatment could be started.

In order for exchange transfusion to be most effective it should be started at the earliest time possible after evaluation of the patient and the circumstances surrounding ingestion of poison permits. Procrastination may result in irreparable damage to vital organs. However, it should be realized that exchange transfusion will be indicated in only a small per cent of patients admitted for acute poisoning.

To facilitate the exchange, we have asked the surgical department to place the polyethylene catheter in a saphenous vein through a femoral cut-down. The usual precautions should be taken, as in transfusion during the newborn period, and at least the estimated blood-volume of the patient exchanged. Prophylactic penicillin and streptomycin post-exchange is an additional routine measure.

SUMMARY

Acute poisoning in service children appears to be at least 2½ times a more common cause for hospitalization in Hawaii than generally expected. A small per cent of admissions had ingested and absorbed sufficient quantities to require more than the usual preventive and supportive treatment. Three of 142

children admitted to Tripler Army Hospital in 1956 and 1957 were treated with exchange transfusion following ingestion and poisoning with large amounts of either Nembutal (brand of pentobarbital sodium), Pyridium (brand of phenylazo-diamino-pyridine hydrochloride) or aspirin. No deaths occurred in this period. Exchange transfusion in selected cases of acute poisoning is effective, and without serious hazards when the technic learned by pediatricians for use in the neonate is practiced, and should be remembered as probably the tool of choice when dialysis is needed.

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MORE DOCTORS—LESS DISEASE?

"We are continually being reminded of the advances and progress of medicine, and unquestionably many things are possible today which were impossible one or two generations ago. But, without detracting from these diagnostic and therapeutic achievements, I think it fair to point out that the real progress is much less than is claimed. We have managed to change the pattern of disease: that is all. As in everything else, solving one problem leads to the appearance of new ones, often more difficult or intractable than the first, and in some directions at least we seem to be approaching problems that cannot be solved. Proportionately, more doctors are kept busy today than fifty or a hundred years ago; but taken as a whole, there seems to be no evidence that there is less disease. Where once a man's first illness might well be his last, today he can clear many obstacles before reaching his grave."

—R. L. MANNING
in *Lancet*
p. 1274, Dec. 21, 1957

PULMONARY VASCULAR CHANGES WITH STENOSIS OF PULMONARY AND TRICUSPID VALVES AND SMALL RIGHT VENTRICLE

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THE possibility of surgical intervention to alter the natural history of some patients with congenital heart disease has stimulated a considerable amount of interest in clinical diagnosis and also in the pathologic changes that occur elsewhere in the body and are directly associated with the cardiac defect. Nature often performs experiments for us that we may utilize as an investigative tool to further our knowledge of some aspects of medicine. For this reason we wish to report an unusual cardiac defect and the pulmonary vascular changes that occurred with it.

REVIEW OF LITERATURE

The arterial vasculature of the lung has been arbitrarily divided into elastic arteries, muscular arteries, and arterioles.[1] The structural changes occurring in these vessels after birth were clearly described by Civin and Edwards[2] as follows:

Elastic arteries. At birth these vessels are patent oval structures, lined by endothelium immediately applied to a wavy internal elastic lamina. This lamina is surrounded by circularly oriented smooth muscle, the external third of which contains scattered elastic fibers. The adventitial portion of the vessel blends with the relatively acellular peribronchial collagenous connective tissue. At birth and shortly thereafter the luminal diameter is equal to or less than the wall thickness. By the first month of life the luminal diameter exceeds the thickness of its wall, the internal elastica becomes heavier, the inner two-thirds of the media is constituted by a mixture of collagenous tissue and smooth muscle, and the outer third demonstrates heavier and more numerous elastic fibers. At six months the vessels resemble the adult structure, particularly with equal distribution of elastic fibers throughout the media; and there is no progressive change in size from one to 20 years.

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Muscular arteries. Prenatally these arteries are represented by circular vessels with very narrow lumens, often with cuboidal endothelial cells lying on a thick muscular media surrounded by a collagenous adventitia that merges with the collagenous structures of the surrounding alveoli. Within the first six months the vessel and its lumen increase in size, the wall does not increase as rapidly as the lumen it surrounds, however, so that at six months the latter is larger than the wall. An internal elastic lamina begins to appear between the fourth and the sixth month but not before the fourth month. It is initially represented by discontinuous strands of elastic fiber that fuse and form a continuous membrane; and the adventitia becomes thinner. Growth is the prominent feature from then to one year. There is the development of an external elastic lamina by two years, and from this time until the age of 20 there is a recapitulation of these changes in other muscular arteries, but past 20 no further differentiation occurs.

Arterioles. These investigators[2] claim that the arterioles are not identifiable prenatally or at birth. At about one month of age the vessels present as small lumens surrounded by a dense connective tissue layer; the developmental changes thereafter consist of progressive increase in luminal size and the formation of a more elastic wall. The time of formation of the elastic lamina was not ascertained, however it was found to be present by the twentieth year.

The progressive vascular changes occurring with age have been studied by Welch and Kinney,[3] and by Civin and Edwards.[2] A systematic progression was noted. In the young individual without pulmonary vascular disease there is little or no fibrous tissue between the endothelium and the internal elastic lamina; in fact, the latter authors stated that they found no intimal fibrosis in the vessels of individuals up to 10 years of age. With the second decade, there was noted asymmetrical intimal involvement principally of the muscular arteries and the arterioles. The third decade carried with it the involvement of the elastic arteries as well. By the fifth decade, there was symmetrical intimal collagenization of the muscular arteries and symmetrical fibrosis of the elastic vessels. Further aging was accompanied by progressively more severe effect on the pulmonary vascular structures, with two generalizations: (1) changes appeared more severe in the smaller than in the larger vessels; and (2) there was an increase in intimal change and increase in the number of vessels involved with increasing age.

It was also noted that as intimal fibrosis became established, fragmentation of the intimal elastic laminas occurred, and the vessel walls showed an increase in the collagenous content with diminution in the elastic and muscular components. Welch and Kinney[3] believed the common factor in the changes that they described was a marked increase in pulmonary blood flow.

Hamilton, Woodbury, and Woods[4] demonstrated that the fetal right and left ventricle pressures are about equal, and it was shown in addition by Lind and Wegelius[5] that in the human fetus the right ventricle supplies blood to the descending aorta via the ductus arteriosus. Civin and Edwards[2] suggested that the high pulmonary resistance necessary for this to occur might not be attributed to the dynamics of the collapsed lung, but seem intrinsic in the pulmonary vasculature, and that the narrow state of the muscular arteries may be the significant factor. Consistent with this are the postnatal changes that occur, principally the widening of their lumina and the thinning of their walls, both of which would lead to a decreased pulmonary resistance to flow.

Edwards,[6] and Edwards and Chamberlin,[7] furthering this line of reasoning, directed attention to the pulmonary vasculature in governing the clinical course of the congenital cardiacs. Other authors cited by Dammann and Ferencz[8] regard the pulmonary changes that occur with cardiac malformation as a separate anomaly of independent origin. This view is based on the lack of correlation in the pulmonary changes with the size of the cardiac defect. Dammann and Ferencz[8] are currently studying this aspect of the problem to attempt to improve the understanding of these particular changes. Edwards[6] has theorized that the vascular changes associated with cardiac defects are a response, a compensatory process, to enable the individual to survive and that these changes are progressive and may become deleterious to the patient.

The particular instance in which we are interested is the defect between the ventricles and the great vessels permitting increased blood pressure to act upon the pulmonary vascular bed. Under these circumstances the pressure applied to the pulmonary circuit is a function of the size of the defect. In all cases, there must be a pulmonary hypertension throughout the life of the individual. The relative blood flow to the greater and lesser circulations is a function of the resistance to flow offered by each. Although the systemic resistance is relatively static, the pulmonary resistance varies during life in accordance with the arterial changes in the lung. It follows that three possible clinical syndromes may occur depending upon the possible changes in the pulmonary arterial system.[9-10]

Phase I. Pulmonary vascular evolution follows a relatively normal progression with a fall in pulmonary vascular resistance. This results in larger amounts of blood being directed to the lungs, and in order to maintain an adequate systemic flow cardiac output increases. Individuals with this condition often terminate in high output failure.

Microscopically, the lungs of an individual dying during phase I show extensive congestion, with edema of the alveolar

septums, and presence of fluid and heart failure cells. The larger vessels show evidence of medial hypertrophy, the smaller muscular arteries show medial hypertrophy with minimal rare intimal proliferation. The capillaries are engorged, and in general there are no venous lesions.

Phase II. The pulmonary vessels remain in the fetal state with thick walls and narrow lumina. In these instances the pulmonary and the systemic resistances may be nearly equal, the patient may manifest few if any signs of cardiac decompensation, and may show little evidence of cardiac disease.

Microscopical examination of the lungs of these individuals fails to demonstrate evidence of failure, but rather the alveoli and capillaries are normal. The small arteries retain their thick walls and narrow lumens, and show increased amounts of muscular and elastic tissue in the walls.

Phase III. Vascular resistance continues to develop in the pulmonary circuit with the result that more blood is shunted to the systemic circulation. Dyspnea and cyanosis are present, growth and development are retarded because of the partially anoxic state.

The increased vascular resistance is caused by marked medial hypertrophy and intimal proliferation. Both small and large vessels are affected. Some small vessels may appear normal; however, serial sectioning usually demonstrates that they lie beyond a point of nearly complete occlusion.

CASE REPORT

The patient, a male infant, was the fourth-born of a 26-year-old Caucasian mother and a 29-year-old Caucasian father. There were three living siblings, no history of familial disease, nor are the parents related by blood. The mother's blood group is 0 Rh positive. Her expected date of confinement was 22 September 1957. The date of delivery was 27 September 1957. Her prenatal course was uneventful, and there was no history of infection or irradiation during pregnancy. Labor and delivery were not remarkable. The length of the first stage was 10 hours, and sedation included Sodium Amytal, Demerol Hydrochloride, and Chlorpromazine Hydrochloride. Anesthesia for the delivery stage was administered by local infiltration, the second stage lasting seven minutes.

Immediately after birth the infant was noted to be cyanotic; however, he responded well and the initial cry was vigorous. No resuscitation was considered necessary, and only bulb suction of the nasopharynx was instituted. Respirations were established in one minute and were considered normal in five minutes. Oxygen was used in the delivery room for 30 minutes. Physical examination at that time revealed an apparently normal newborn baby. The initial cyanosis was

thought by the attending physician to be the result of amytal depression. It was decided at this point to place the infant in an Isolette with 35 per cent oxygen. The nursing notes indicated that this treatment improved his color slightly and that he seemed active and alert.

On the first day after birth the infant was removed from the Isolette. Cyanosis was noted to persist and to become worse. Pulse rate at the time was 120. Examination of the lungs was not remarkable. A grade iii harsh systolic murmur was heard over the lower sternum and P-2 was diminished, but no hepatosplenomegaly was noted. The attending physician entertained the diagnosis of congenital heart disease. A reoentgenogram of the chest was thought to indicate a normal newborn chest.

During the first day of life, the pulse rate increased to 134 per minute and respirations to 42 per minute. The cyanosis was persistent, but the infant's condition otherwise remained unchanged. By 25 hours after birth, he was noted to be retracting, respirations had increased to 50 per minute, and there was marked cyanosis on slight exertion. By 36 hours after birth, cardiac enlargement was noted, the liver edge was 2 cm below the right costal margin, and 2 to 3 plus pretibial edema had developed.

It was decided to digitalize the patient after an electrocardiograph was obtained that demonstrated left ventricular hypertrophy. A clinical diagnosis of tricuspid atresia with patent foramen ovale was made. By 50 hours after birth, the pulse rate had increased to 160 per minute and respirations to 60 per minute. Cyanosis had increased and the child's condition was considered serious. By 62 hours after birth, the cyanosis was marked with crying, respirations were labored at 52 to 60 per minute, and the pulse rate persisted at about 160 per minute, despite digitalization. Physical examination revealed the lungs to be clear to percussion and auscultation, the heart border extended to the left anterior axillary line, the second pulmonic sound was diminished, and the previously mentioned murmur persisted. The liver edge had descended to 3 cm below the right costal margin, and peripheral edema was still noted. Penicillin and streptomycin were given prophylactically.

The infant's condition deteriorated. Periods of apnea lasting as long as 70 seconds were noted approximately 60 hours after birth. By this time the pulse rate had increased to 184 per minute, and respirations to 72 per minute. The infant was cyanotic and breathing irregularly. In the terminal three hours the pulse rate dropped from 180 per minute to 120 per minute. Periods of apnea became longer, and Cheyne-Stokes respiration was noted two hours before death. Terminally, the child was gasping and markedly cyanotic.

During hospitalization, no fever was noted. Birth weight was seven pounds and twelve ounces, and terminal weight was seven pounds. Death occurred 72½ hours after birth.

A postmortem examination was performed about 13 hours after death. Significant findings included changes in the heart, lungs, and liver. The lungs filled the pleural cavity and there was no evidence of free fluid, exudate, or hemorrhage. The left lung weighed 22 grams; its surface was smooth and glistening, the parenchyma was light red-purple in color, and the left lower lobe showed a deep red-purple area in the dependent portion. The entire lung was crepitant and floated on water, the cut surface being light red-purple. The vascular structures supplying the lung were in their usual distribution. The right lung weighed 29 grams and was similar in description to the left.

The heart was enlarged, weighed 21 grams, and extended from 0.5 cm to the right of the sternal border to the anterior axillary line on the left. The pericardial cavity contained about 5 ml of deep golden-yellow fluid; the surface of the heart was gray and glistening, through which a deep red-purple parenchyma was noted. The right atrium was tremendously enlarged and dilated. The surface area occupied by the right ventricle was small.

Dissection of the vessels at the base of the heart revealed them to rise from their appropriate cavities. The ductus arteriosus was located approximately 3 cm distal to the aortic ring. The right atrial wall measured 3 mm in width. The foramen ovale was patent between the atrial cavities, and measured 1.5 cm in diameter. The tricuspid valve viewed superiorly represented an inverted cone. The valve ring at the base of the cone measured 2.5 cm in circumference. At the apex of the cone, the valve orifice was represented by a slit 0.6 cm in length and 0.1 cm in width. The valve leaflets were well formed and although they were fused along their margins, the line of junction was discernible. The chordae tendineae inserted immediately above the valve edge. The small slit-like aperture described above formed a stenotic point in the circulatory path.

The right ventricle was represented by a small, underdeveloped cavity, its wall 0.6 cm in width and its endocardial surfaces smooth, shiny, and translucent. The papillary muscles were large and the chordae tendineae well formed. The interventricular septum demonstrated no persistent interventricular communication.

There was no evidence of infundibular stenosis. The right ventricular out-flow tract was essentially closed by a stenotic pulmonic valve. This structure was represented by a dome-shaped fusion of the valve leaflets. The circumference of the pulmonic valve ring was 1.0 cm. The leaflets were well formed and the line of fusion between adjacent cusps was evident. The orifice was situated at the highest point of the dome, and was a tiny slit-like orifice that would just admit the tip of a needle. Approximately 1 cm above the pulmonic valve ring, the pulmonic artery was circumferentially narrowed, so that its lumen had a diameter of about 0.5 cm. The lumen before and after that portion, however, was 1- to 1.2 cm in diameter.

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The left atrium was not remarkable except for the patent foramen ovale previously noted. The left ventricular cavity was not remarkable. The left ventricular wall measured 0.9 cm in width. The mitral valve circumference was 4 cm. The aortic valve circumference was 2 cm, and the coronary vessels were not remarkable. The ductus arteriosus previously mentioned was 1 cm in length. The venous system of the surface of the heart and the coronary sinus were remarkably dilated.

Careful dissection of the arch of the aorta failed to reveal any further abnormality. The liver was enlarged, weighed 360 grams, extended 2.5 cm below the right costal margin. The capsule was smooth and shiny, through which a red-purple parenchyma was noted. Aside from passive visceral congestion, the rest of the postmortem examination was not remarkable save for a solitary frontal-lobe brain cyst.

Microscopic examination of the liver and kidneys demonstrated congestion; and of the brain, demonstrated a cyst, the margins of which failed to show evidence of gliosis or inflammatory infiltrate.

Microscopic examination of the lungs was of particular interest. Many of the alveolar spaces were filled with an eosinophilic granular material containing scattered hemosiderin-laden histiocytes. There were large confluent alveolar spaces, the walls of which were formed by hypercellular septums. There were areas, particularly in the upper lobes bilaterally, that demonstrated failure of expansion; in these areas the alveolar walls were collapsed and adjacent. In other areas the alveolar septums themselves were edematous. The alveolar capillaries were markedly distended with blood.

The bronchial and bronchiolar structures of the lung were not remarkable, but the pulmonary vascular tree demonstrated changes we would like to describe in detail. In examination of the lung tissue, making use of the routine hematoxylin and eosin stain and also Verhoeff-van Gieson's and Gallego's elastic-tissue stains[11] we observed the following.

Elastic arteries. The larger elastic arteries accompanying the primary bronchial division were, in general, luminally as wide as the air passages they accompanied. The adventitial portion was composed of collagenous connective tissue. The media was formed of elastic and muscular tissue, the elastic fibers were nearly evenly distributed throughout the medial portion and were well formed. The internal elastic lamina was a prominent, rather smooth instead of wavy, structure. We also were able to demonstrate areas of fragmentation of this structure. There were asymmetrically placed areas of intimal fibrosis (fig. 1) lying above the internal elastic membrane, and often above the areas of fragmentation (fig. 2). The endothelium was represented by a thin line of flattened cells.

In the smaller elastic arteries, the over-all diameters were still equal to the air passage they lay near. The intimal and medial changes

Figure 1. Photomicrograph of an elastic artery showing a plaque of intimal fibrosis with fragmentation of the internal elastic lamina. (van Gieson's elastic tissue stain, × 440)

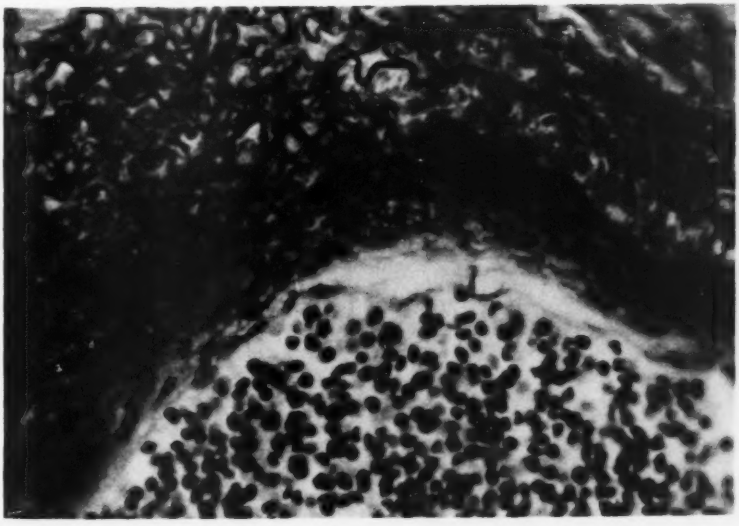

Figure 2. Photomicrograph of an elastic artery showing fragmentation of the internal elastic lamina. (van Gieson's elastic tissue stain, × 440)

noted in the larger vessels were present in these structures also, but more infrequently. These vessels demonstrated a cuboidal endothelium lying upon a wavy internal elastic lamina with a predominance of elastic tissue fibers in the outer third of the media. Even in these structures, however, the media was thicker than one would anticipate and the lumen larger (figs. 3, 4).

Muscular arteries. The muscular arteries were circular in cross-section, and in general their luminal diameter exceeded the thickness of the wall including the intima, media and adventitia (fig. 5). Often the lumen exceeded twice the thickness of the wall. The intimal lining varied from a cuboidal to a flattened cellular structure lying upon a heavy muscular media. In about 20 per cent of these vessels a ring formation of an internal elastic lamina could be demonstrated by Gallego's stain. (We were unable to demonstrate this photographically on black and white film because of the hue differences in blue and purple.) In general, the luminal diameter of these vessels was less than that of the nearest bronchiole. In H and E stained sections, an eosinophilic "smudging" was noted along the intimal linings.

Arterioles. Arterioles were identified by the adventitial collagenous connective tissue that surrounded the small lumen. There was virtually no muscular or elastic tissue within their walls. These vessels were patent and their luminal diameters were less than the wall width (fig. 6).

DISCUSSION

The mechanism of formation of this or any particular congenital defect is not known. However, the general association of right ventricular under-development, tricuspid valve anomaly, and pulmonic valve failure of development is a commonly occurring triad.[12] The particular instance that we are reporting included right ventricular under-development, tricuspid stenosis with but a minute orifice, pinpoint valvular pulmonic stenosis, widely patent foramen ovale, patent ductus arteriosus and a well developed pulmonic artery with an area of post-valvular stenosis presenting a demarcated circumferential narrowing a short distance from the stenotic pulmonic valve. It is interesting to note that the diameter of the interauricular communication was greater than that of the pulmonary artery at its widest point, 1.0 cm, and of the aorta at its origin, 0.7 to 0.8 cm.

It would seem that the prenatal circulation was as follows: Blood returned via the vena cavae entered the right auricle and passed via the foramen ovale to the left auricle, thence to the left ventricle and to the aorta. Considering the stenotic pulmonic valve, the tricuspid stenosis, and the under-developed right ventricle, little blood-flow would be supplied via this route to the pulmonary bed. In fact, the widely dilated ductus arteriosus would expose the pulmonary vasculature to systemic pressure. In other words, the systemic and pulmonary circuits were supplied

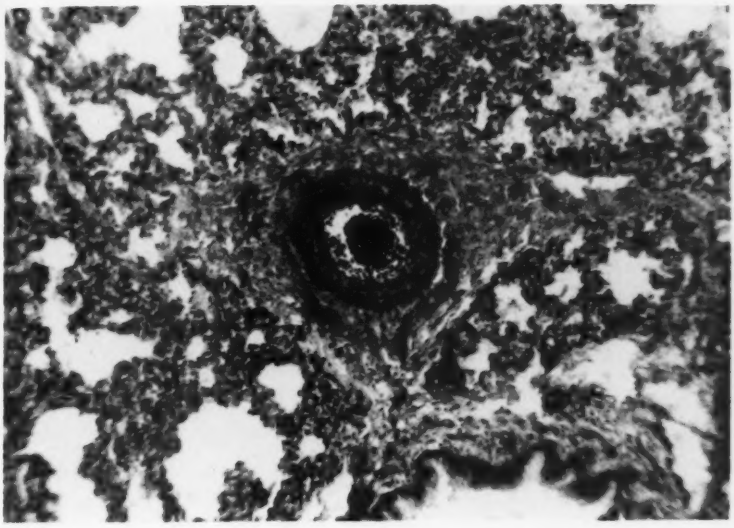

Figure 3. Photomicrograph of an elastic artery, well formed, but with increased amount of elastic tissue. (van Gieson's elastic tissue stain, × 440)

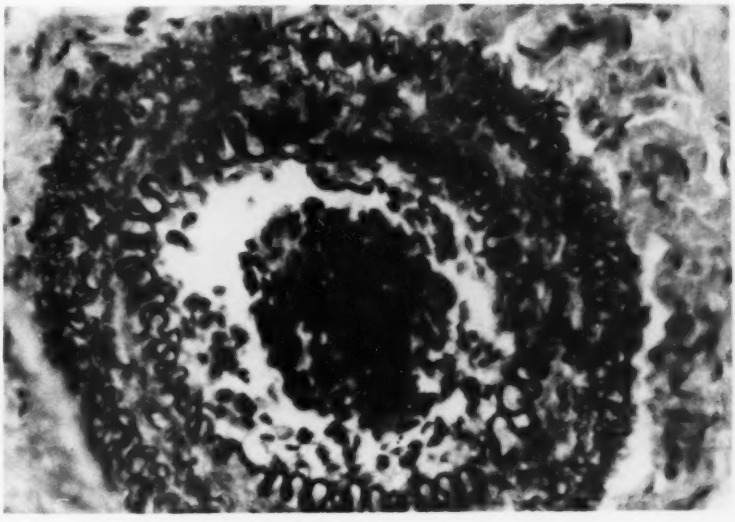

Figure 4. Photomicrograph of same field as in figure 3. (× 400)

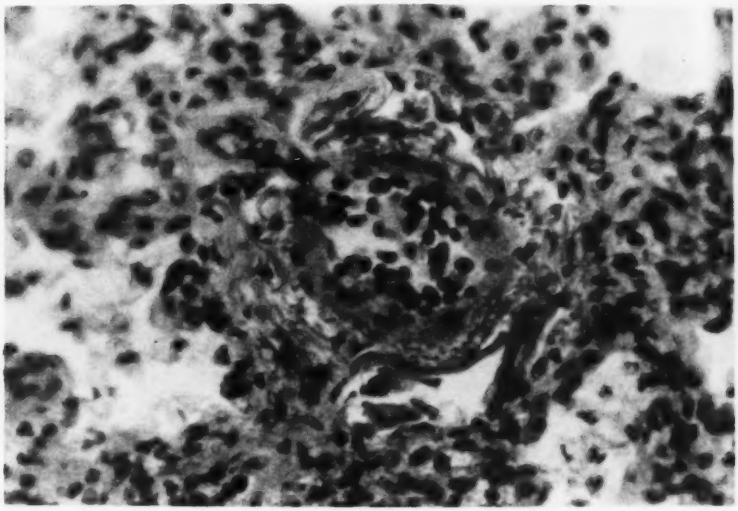

Figure 5. Photomicrograph of muscular artery showing a large circular lumen, the diameter of which exceeded the wall width. (van Gieson's elastic tissue stain, × 400)

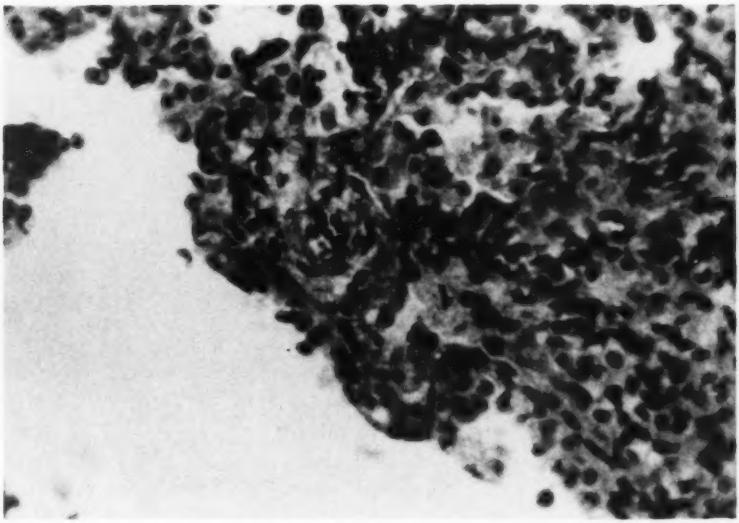

Figure 6. Photomicrograph; a single arteriole is shown at the middle of the field, about 2 cell widths from the air space. (van Gieson's stain, × 440)

by a common ejectile force as mentioned by Edwards, Edwards and Chamberlin.[6,7] Postnatal circulation was probably identical to prenatal circulation. One can only speculate as to the volume of blood directed to the pulmonary circuit in view of these circumstances.

We believe that this child died in high output cardiac failure in an attempt to compensate for the poor aeration of blood, teleologically speaking. Dammann and Ferencz[8] have postulated three possible courses or clinical syndromes for individuals with this type of cardiac anomaly. They theorize the progressive fall in peripheral vascular resistance in the pulmonary bed, with a larger and larger portion of the cardiac output being directed in this manner. Cardiac output rises in an attempt to compensate for this loss, and high output failure frequently ensues. It seems to us that some features of this infant's condition fitted the picture of phase I, but the pathologic changes of the pulmonary vasculature, notably the elastic arteries, were more marked than those reported.

We call attention to this case because of the rather marked vascular changes, particularly in the larger elastic arteries, which would be exposed to the highest pressure gradient. These, including intimal proliferation and fragmentation of the internal lamina, are considered by many to be early degenerative changes. We find this of decided significance in view of the fact that this patient was only three days old at the time of death. We know of no previous report of so marked vascular change in such a young infant. On the basis of the work done by Civin and Edwards,[2] by Dammann and Ferencz,[8] and by Welch and Kinney,[3] these vessels demonstrate age-changes compatible with six or more months of life.

The apparent rapid "aging" of these vessels raises an interesting point. Fetal ventricular pressures are about equal, but blood flow occurs from the pulmonary artery via the ductus arteriosus to the aorta. This route presupposes a higher pressure at the pulmonary end of the ductus, and therefore in the pulmonary artery, than in the aorta at the aperture of the ductus. The best explanation for this phenomenon includes competency of the pulmonic and aortic valves, and high pulmonary resistance—either on the basis of a collapsed lung phenomenon or the intrinsic vascular structure—and a lower systemic pressure. Blood in the pulmonary artery and aorta at the moment of valve closure must be equal; however, the rapid "run off" in the systemic bed would result in the higher pulmonary artery pressure necessary for this to occur. Total systemic pressure is the sum of the right and left ventricular pressures, assuming no obstruction to flow.

The child we discussed must have had a systemic pressure derived only from the left ventricular pressure component. There must have been a reversal of flow through the ductus arteriosus and bronchial arteries in order to fill the pulmonary vessels in the manner found at postmortem.

Although this is pure conjecture, the mature nature of these pulmonary vessels may be explicable in part by assuming that these vessels were subjected to an increased pressure prenatally, and may in fact have developed more in the manner of systemic than of pulmonic arteries. The changes seem too marked to have occurred solely in the short postnatal life. Unfortunately, we have neither the material nor the facilities to subject this to experimental proof.

SUMMARY

A brief review of the literature on pulmonary vascular changes in congenital heart disease, together with the case history of an infant with an unusual group of cardiac anomalies and rapid termination, are reported. Attention is directed to the rapid "aging" of the arteries in the pulmonic circuit. In view of the short life span of the infant, these changes are significant and more severe than any previously described by other authors.

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TEAMWORK IN MEDICAL RESEARCH

"The quality of medical care rests upon the quality of medical education as related to research. The organization and endowment of medical research, the establishment of institutes for scientific research, and the multiplication of opportunities to advance knowledge and specialize has enabled large numbers of young men who otherwise could not afford to give their whole time to research and teaching, to do so without distracting anxiety. Today, more than ever before, generous endowments and fluid funds have been made available by the public, either through voluntary giving or by means of tax funds, so that opportunities for original work have greatly increased. Formerly, researches advancing medical knowledge and medical practice were largely due to individual and isolated effort but now teamwork and group enterprise has become more general and prolific. As the late Francis Blake remarked when he was President of the Association of American Physicians, the big change in medical communication in the past 50 years has been in the production of research and writing by multiple authorship rather than by individuals."

—CHESTER S. KEEFER, M. D.
in *The American Journal of Gastroenterology*
p. 196, Feb. 1958

THE ROLE OF ELECTIVE PLASTIC SURGERY IN MILITARY HOSPITALS

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PLASTIC SURGEONS in the Armed Forces today are besieged with requests for elective surgical procedures. It frequently is difficult to decide what is to be considered strictly elective surgery. There is no question that a large part of plastic surgery is elective. Revision of scars, hand reconstruction, correction of burn deformities, reconstruction after carcinoma ablation, and a great many other procedures, exclusive of acute trauma, are technically elective; nonetheless, many of these are necessary operations, from both a functional and an esthetic viewpoint. It behooves the reconstructive surgeon to define criteria for cases to be selected for operation.

It is apparent that such operative procedures as rhinoplasty, otoplasty, mammaplasty, dermabrasion, and others, if desired primarily for the sake of appearance, must be carefully and individually considered. The decision must be based on the merits of the individual case.

A purely cosmetic operation, in the strictest sense, is difficult to defend in the Armed Forces. Such an operation may mean many man-hours lost, unnecessary utilization of hospital beds, and possible involvement in medicolegal problems that are difficult to solve. In addition, it does not enhance relations with civilian colleagues in this field of work.

It must be remembered that any surgical procedure can result in complications. Whenever a surgeon lifts his scalpel to create a surgical wound, he is opening the door to the possibility of complications. This holds true no matter how trivial the surgical procedure may be. Military surgeons performing elective surgery must be always conscious of this possibility, and remember the dictum: "There is no such thing as a 'minor' operation." Complications can be of a minor nature or can result directly or indirectly in death. In a patient sensitive to local anesthetic, a very few ml can result in a serious or fatal reaction. Infection, hemorrhage, wound dehiscence, skin slough, thrombosis, embolism,

From U. S. Naval Hospital, St. Albans, N. Y.

and other surgical complications occur regularly, although not frequently, despite careful adherence to surgical principles. Every operation is a gamble with nature, the odds depending on the operation to be performed, the skill of the surgeon, postoperative care, and to a certain extent, on individual variation.

For practical reasons, the remainder of this article will concern itself with only those operative procedures that can properly be termed 'purely elective' or cosmetic (an unfortunate word). Elective procedures involving scar revision, cancer excision and reconstruction, hand surgery, burns, traumatic deformities of the extremities, maxillofacial procedures, et cetera, are those most often clearly indicated in service personnel. The possible exception is the patient with a physical deformity that existed prior to his entering military service. This will be commented on later. In this discussion, purely elective or cosmetic plastic surgery refers to rhinoplasty, mammaplasty, dermabrasion, myeloplasty, and correction of prognathism (without malocclusion) and of certain congenital deformities or the results of their repair, *i. e.*, secondary harelip deformity and hemangioma.

Congenital anomalies lie somewhere between the two broad categories of necessary and elective surgery. Under the present laws, a child born with a congenital anomaly such as a cleft lip, cleft palate, or syndactyly, can be treated in service hospitals or by a civilian surgeon under the Medicare program. Most congenital deformities can hardly be considered as purely elective. A deformed child is a handicapped child. In most cases, tragic emotional sequelae and functional deformity can be prevented by proper early treatment. These cases are justifiably treated at the earliest possible age, consistent with good judgment. Any other attitude is indefensible.

It is well recognized in plastic surgical departments of the Armed Forces, that the lay public of today is cosmetic conscious. Motion pictures, lay publications, and the entire advertising industry of America constantly paint a picture of the desirability of physical perfection and beauty. Motion picture stars are national symbols of this orientation, and this consciousness reaches everyone to some degree. Many articles appear in lay publications, extolling the virtues of cosmetic surgery. The result is an increasing demand from service personnel and dependents for elective cosmetic surgery. The plastic surgeon is literally barraged with requests for this type of surgery. Once he undertakes a correction of a "hump nose" or protruding ears, the word is quickly spread in his military area, and the stampede is on.

Elective plastic surgery has a definite place in a military hospital. The cases must be carefully selected on individual merit, after a thorough evaluation. At this point let us cite a hypothetical case of the type the military plastic surgeon sees with some regularity. A serviceman presents himself requesting

a rhinoplasty for a large "hooked" nose. On interviewing the patient, the information is brought out that he is a career serviceman who has done a good job in the past, and been promoted on schedule. He intends to remain in the service as a career.

From the past record of the patient, the indications are that he is a valuable man who will be an asset to his unit and to the service in general. It is further brought out that he suffers from severe feelings of inferiority, frequently made worse by good-natured but misdirected chiding from his friends. Self-consciousness of his grotesque nose increases, and he may find himself withdrawing from social events. He may feel inadequate around members of the opposite sex, often withdrawing from "dates" altogether. This mental fixation on his nose can increase to the point of severe emotional disturbance, in an otherwise healthy individual. It may interfere with his work efficiency, retard his career advancement, and affect natural social growth.

To retain valuable career personnel, every effort should be made to prevent or deter factors, either physical or mental, that might be detrimental to individual career advancement. To an enlisted man, the cost of an elective nasal operation in a civilian hospital often is prohibitive, and the only possibility for an operation, and coincident mental rehabilitation, is by elective surgery in a service hospital. Such a case should be carefully considered for rhinoplasty, which is justified, barring legitimate contradictions. It is a common experience to find increased effectiveness in work and attitude in such a patient, all adding up to a happier, and thus more valuable, individual.

Nasal deformities often are associated with faulty air passages resulting from internal derangement or deviated nasal septum, which may be a predisposing factor in sinusitis.[1] It is current practice in these cases to do a combined submucous resection and rhinoplasty (fig. 1). The cases with obstructed nose constitute a separate category, and there is little or no contraindication to doing corrective nasal surgery on such patients.[2] The indication for elective operation here is clear-cut.

Another deformity with strong emotional overtones is protruding ears or "bat ears." Most parents today are aware of the possibilities of correction of protruding ears in their children and seek operation. It is our belief that no child should be denied correction of "bat ears." The emotional sequelae of this condition can be severe in young adulthood.[3] Young children tend to be merciless with their comrades, thus the common nicknames "Dumbo," "Mickey Mouse," and "airplane ears." It is preferable to correct protruding ears at about four years of age, before school begins and after full ear growth has occurred (fig. 2). The operative correction is simple and need not involve more than overnight hospitalization.[4] This deformity should properly be considered congenital, and technically does not fall into the "cosmetic" category.

Figure 1. A 24-year-old career serviceman with an elongated, externally deviated nose that was partially obstructed as a result of severe septal deviation. This deformity was of considerable emotional concern to the patient. Rhinoplasty and submucous resection in one operation resulted in complete correction of the airway obstruction and a pleasing cosmetic effect. Technically an elective operation, it obviously was necessary both to provide proper nasal ventilation and to improve the patient's morale.

Figure 2. A service dependent with typical congenital protruding ears. The deformity was corrected by a simple operation, involving only a 3-day hospitalization, performed at 5 years of age after full ear growth. In the lateral views, note reconstruction of the antehelix fold at time of operation.

Plastic operations on the female breast are rarely indicated in military hospitals. So-called augmentation mammaplasty or breast "build-ups" have attained certain vogue in civilian practice. This procedure increases the size of the breast in deficient cases, and consists of the implantation of autogenous dermis-fat grafts as pedicles or free grafts, or of the implantation of a

prosthetic polyvinyl sponge. In most cases, this is purely a cosmetic procedure, but it can be attended by the complications of major surgery; in the case of polyvinyl sponge, there may be excess hardening and shrinkage resulting from fibrous invasion of the material.[5] It is understandably not a procedure that is justified in military practice. Occasionally, dermis-fat grafts are warranted after simple mastectomy for benign disease. The removal of a breast is a severe emotional handicap to a woman. In some instances it is possible to reconstruct the mammary eminence with local flaps, after removal of all underlying breast-tissue in benign lesions.

The other form of mammaplasty is breast reduction for overlarge, or hypertrophic breasts. In massive mammary hypertrophy or gigantomastia, definite pathology can result.[6] Kyphosis, cervical and dorsal spine arthritis, and grooving of the shoulders, all can result from excessively overweight breasts.[7] This is analogous to a woman carrying a heavy sack of sugar suspended from each shoulder. Under these conditions, a mammary-reduction operation is a necessity (fig. 3). Again, each case must be considered on individual merit.

The technic of dermabrasion, or surgical abrasion of the superficial skin layers, is widely practiced today. This technic was developed during World War II for removal of imbedded dirt and debris in wounds. It since has been adapted for the ablation or camouflage of acne scars, decorative tattoos, benign lentiginous spots, moles, and other benign skin lesions.[8] It most commonly is sought after by victims of acne-scarring of the face. The operation is simple, and merely involves the abrasion of superficial skin by sandpaper, wire brushes, or similar devices. Morbidity is low, and mortality negligible. In the military, it is indicated for treatment of traumatic tattoos or scars, and to improve healing of wound edges after scar excisions. Acne cases must be carefully selected.

The number of decorative tattoos in military personnel is legion. Tattoos often are acquired while the individual is intoxicated. Self-decoration is thought to be related to "attention-getting mechanisms" or possibly the desire for self-assertion. Frequently, the young serviceman regrets his action almost immediately. By and large, the removal of tattoos from all those who desire it is impossible; there are not enough beds, operating rooms, surgeons, or hours in the working day. It also must be remembered that the removal of a tattoo frequently involves skin-grafting, a major operation with the inherent danger of complications such as partial loss of the skin graft and infection. There are certain instances, however, when it may be considered. A patient who has been tattooed while stuporous, it having been arranged for by well-meaning but misled comrades, is a candidate for operative removal. Certain religions ban this sort of decoration, and in

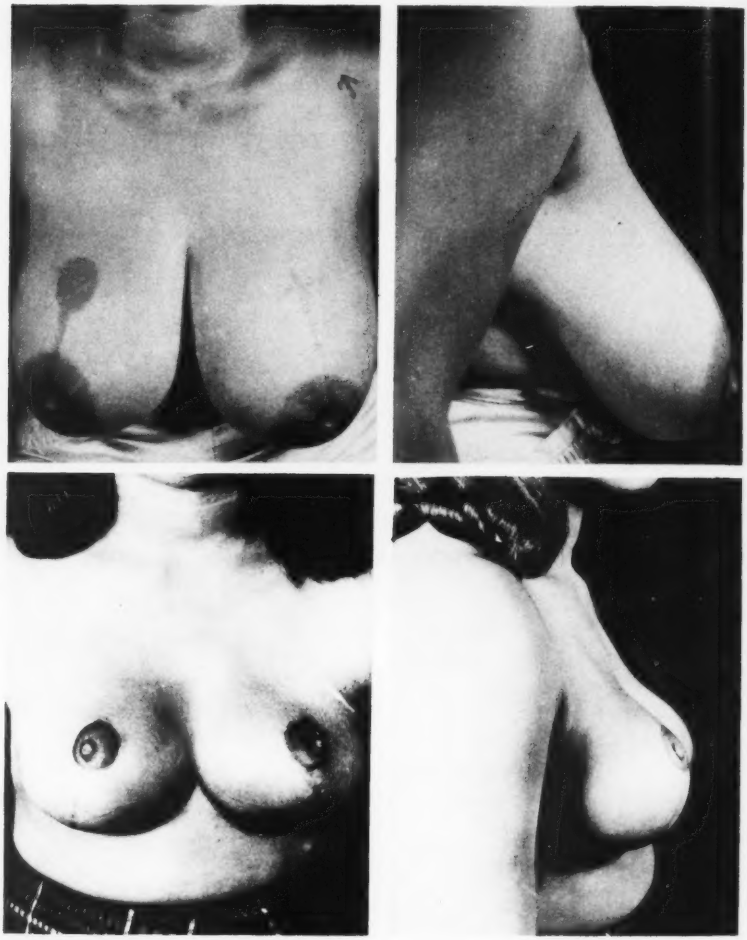

Figure 3. Painful gigantomastia in a 23-year-old service dependent. An example of virginal hypertrophy, these breasts had been heavy and painful since puberty. Note the deep grooves in the shoulder caused by brassiere straps. The dye markings on the skin represent preoperative planning of nipple sites. Bilateral mammaplasty, performed in one operation according to the McIndoe modification of the Biesenberger technic, included removal of 5½ to 6 lbs of tissue from each breast. The patient was greatly relieved by this procedure, and noted disappearance of formerly chronic low back pain, undoubtedly of mechanical nature.

these instances the benefit of the doubt should be given the individual.[9] Another possible indication for surgical removal of tattoos is in the instance of lewd or vulgar tattoos in career personnel. A distasteful tattoo may be a permanent handicap to

career advancement. Traumatic tattoos are those in which dirt or debris has been ground into a wound at the time of trauma. In service-connected traumatic cases, the removal, by dermabrasion or excision of tattooed scars is unquestionably justifiable.

When a purely elective case is selected for operation, the patient must of necessity be placed low on the priority list for a hospital bed. Each plastic service has its own system for scheduling patients. Hand reconstruction, trauma, burns, cancer, and major reconstructive work stand at the head of the priority list. Elective surgery must be scheduled when convenient. The number of cases selected for operation also must depend on the technical personnel available in a given installation and on the time and bed space available.

In military practice, few patients are treated on an ambulatory basis. A patient must be "fit for duty" before being discharged from the hospital, which involves longer periods of hospitalization and is a factor in planning the elective surgery schedule. Another possible factor influencing the type and amount of elective surgery is the existence of a residency training program. When a hospital of the Armed Forces undertakes to set up a residency in a specialty—plastic surgery in this case—it also undertakes the responsibility of turning out a well-trained specialist. The plastic surgeon must be thoroughly trained in all aspects of his specialty. Elective surgery is a large part of plastic surgery, and it cannot be denied that cosmetic surgery is an important part of the field.

If any doubt arises as to the patient's emotional reactions or motivations regarding an elective operation, it is mandatory to have a psychiatric opinion. In military hospitals, psychiatric consultations are facilitated. An hour-long interview with a psychiatrist frequently will bring out emotional disorders or personality defects that absolutely contraindicate purely elective surgery.

SUMMARY

In considering the role of elective plastic surgery in military hospitals, it is not feasible to establish a fixed set of rules governing acceptance of cases for operation. In general, patients should be selected on an individual basis, after thorough investigation as to psychiatric motivation, value of the individual as a career serviceman, degree of grotesqueness of the deformity, and coexisting anatomic deformities such as a deviated septum.

Congenital deformities most often clearly deserve surgical repair. Nasal plastic surgery should be performed when deformity coexists with airway obstruction or resulted from service-connected trauma, or when there is excessive grotesqueness in size or shape of the nose. Mammary reduction is indicated when the breasts are so excessive in size as to lead to pathologic changes. An evaluation of the facilities, bed space, and individual service

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load should be made, and elective surgery categorized as to priority.

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COURTESY IN THE OPERATING ROOM

"Good manners are important everywhere in the hospital. In the operating room they are a necessity. There is no place and no other time when the surgical patient has more at stake than during the relatively short period he spends in the operating suite. The exercise of correct conduct by medical and nursing personnel facilitates good teamwork and offers proper protection to the patient during these critical moments.

"Displays of bad temper ill become any physician, but they are entirely out of place in the operating room. Teamwork can be destroyed by tongue lashings, use of improper language, throwing instruments, constant complaining and undue sarcasm. Excessive good humor, particularly when accompanied by loud laughter and horseplay, breed irreverence and are apt to led to distraction, time wasting, forgetfulness, and perhaps more serious errors."

—ALLAN J. RYAN, M. D.
in *Connecticut State Medical Journal*
p. 821, Sept. 1957

THE MANAGEMENT OF LATE PROBLEMS FOLLOWING PEDIATRIC CARDIOVASCULAR SURGERY

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NICHOLAS G. BOTTIGLIERI, *Major, MC, USA*

WITH advances in the diagnosis and treatment of congenital cardiovascular anomalies, more and more children with these conditions are now enjoying useful existences far beyond the life expectancy of 15 to 20 years ago. As a result of this, however, physicians are being faced with new problems in the late postoperative care of such patients.

The purpose of this article is to describe the experiences encountered during the care of three patients at an Army hospital some 6,000 miles from the continental United States. All presented recurring problems of management, either related to their original cardiac defects, or as the result of artificial shunts created by the surgical procedure. The cases to be presented were all different in regard to the basic cardiac pathology, but some of the functional problems were somewhat similar. Recognition of the existing functional pathology, as well as the awareness of impending difficulty in patients whose cardiac anatomy is disrupted by a congenital deformity or by surgery, seems most important in supporting good health for these children.

CASE REPORTS

Case 1. This patient was a seven-month-old Caucasian female infant who had undergone surgical intervention for a vascular ring at age three and one-half months while living in the continental United States. The anterior limb of a double aortic arch and a patent ductus arteriosus were ligated.

She was first seen two days after arrival in this command. She had been manifesting symptoms of an upper respiratory infection for the previous two weeks. There was no remarkable temperature elevation, but there were physical signs and roentgenographic evidence of pneu-

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monia at the base of the right lung field. On admission to this hospital a white blood cell count was 11,100 per μl with 27 per cent neutrophils and 69 per cent lymphocytes. Throat culture was negative. An electrocardiogram was within normal limits.

The infant was given 300 mg (300,000 units) of procaine penicillin G intramuscularly each day for five days with complete clearing of the previously described rhonchi in the right lung field. A follow-up roentgenogram of the chest in one week showed complete clearing of the infiltration at the right base. After this episode there was periodic stridor in the recumbent position.

On another occasion the infant developed a streptococcic sore throat and purulent otitis media caused by hemolytic *Micrococcus pyogenes* var. *aureus (Staphylococcus aureus)* organisms. These conditions responded rapidly and satisfactorily to a 10-day course of procaine penicillin G, 300 mg given intramuscularly each day, together with Terramycin (brand of oxytetracycline) administered in the form of ear drops. Subsequent fluoroscopic studies with barium by mouth revealed an indentation of the posterior aspect of the esophagus by the posterior aortic arch and a persisting narrowing of the lower trachea. Over a 10-month period there was a gradual diminution in the frequency of stridor. At present, growth and development are normal. The parents have been instructed to bring the child to the clinic at the earliest sign of respiratory infection.

Case 2. An eight-year-old Caucasian girl was seen in the outpatient department of this hospital on several occasions during the period from November 1956 through March 1957 for frequent upper respiratory infections and one episode of bronchopneumonia.

She had arrived at this command three and one-half years following a right Blalock-Taussig procedure for tetralogy of Fallot. Because of her small stature, congenital scoliosis, and a wide bridge of the nose, she had also been studied for Bonnevie-Ullrich syndrome of ovarian agenesis and congenital anomalies. However, the urinary follicle stimulating hormone excretion was normal. Previous roentgenograms of the chest had shown increased right pulmonary vascularity.

On 20 March 1957 the child was given a course of penicillin for pharyngotonsillitis. On 26 March 1957 she developed a fever of 102°F, a diffusely erythematous urticarial eruption, and one episode of transitory respiratory distress.

Physical examination revealed slight cyanosis of the lips and nail beds, a diffusely red pharynx, fine moist rales in the right lower lobe posteriorly, and a loud systolic left parasternal murmur, most marked in the fourth left intercostal space, transmitted throughout the precordium. The right radial pulse was not palpable and no blood pressure could be obtained in the right arm. The blood pressure in the left arm was 80/50 mm Hg. There was marked sinus tachycardia of 160 per minute. The patient was orthopneic and the veins on the dorsum

of her left hand failed to collapse until elevated 20 cm above the level of the atria. Weight was 33 pounds.

At that time the white blood cell count was 17,050 per μl with 83 per cent neutrophils. The hemoglobin was 15 grams per 100 ml. A red blood cell count was 5.1 million per μl. A roentgenogram of the chest revealed only an increased right pulmonary vascularity (fig. 1). An electrocardiogram showed right ventricular preponderance.

Figure 1 (case 2). Roentgenogram showing increased vascularity of the right lung field.

Because of the persistent tachycardia over a period of one and one-half hours and clinical evidence of left ventricular failure, the patient was digitalized with 0.25 mg of digoxin, every 12 hours for four doses, with a maintenance dose of 0.25 mg each day. The symptoms of failure cleared within 36 hours after admission. On the third hospital day an additional soft to-and-fro murmur arising from the shunt was heard in the right subclavicular and right interscapular areas. The patient was discharged on the seventh hospital day with maintenance digoxin, 0.25 mg each day. During a 10-month follow-up, she has had no major respiratory infections and has no dyspnea on normal activity.

Case 3. A three-year-old Caucasian boy with symptoms of a respiratory infection was first seen shortly after arrival at this command. At 10 months of age he had undergone surgery at Brooke Army Medical Center where a Blalock procedure for tricuspid atresia was accomplished. Preoperative diagnosis had been established by the presence of cyanosis, electrocardiographic evidence of left ventricular hypertrophy, and an angiogram which showed direct passage of the dye from the right to the left atrium. At operation the right subclavian artery was anastomosed to the pulmonary artery just proximal to its bifurcation.

On physical examination there was moderate tachypnea and cyanosis of the lips and nail beds, which increased on crying. A loud systolic murmur was heard best in the third left intercostal space, transmitted throughout the left precordium. There was left ventricular enlargement to percussion. The liver edge was sharp and moderately tender on palpation one fingersbreadth below the right costal margin. A roentgenogram of the chest revealed transverse cardiac enlargement and evidence of bilateral pneumonitis (fig. 2). The white blood cells numbered 11,750 per μl, with 66 per cent neutrophils.

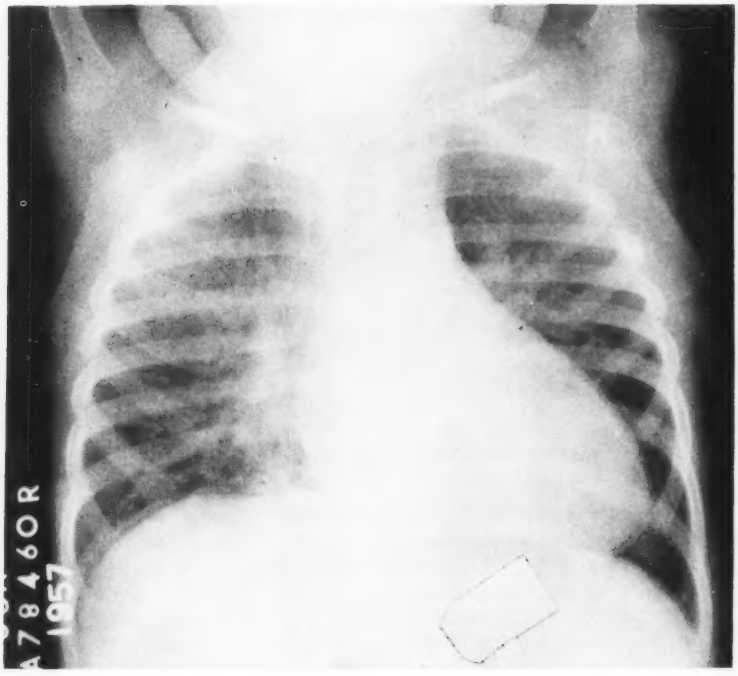

Figure 2 (case 3). Evidence of left ventricular enlargement and pneumonitis in the right lower lung field.

The patient was treated with Terramycin (brand of oxytetracycline) and on examination four days later the lung fields were clear on percussion and auscultation. The apical rate was down to 80 per minute and the liver was barely palpable at the costal margin, descending one fingersbreadth on deep inspiration. Three weeks later, an additional soft systolic and diastolic murmur was heard below the right clavicle, transmitted to the back just medial to the right scapula. This was thought to arise from the site of the anastomosis. Antibiotics were discontinued at that time without recurrence of original symptoms. Since this episode, the patient has been well and is being followed at three-month intervals. He has had occasional slight dyspnea but no orthopnea. He does develop facial cyanosis on crying.

DISCUSSION

The problems presented here are centered around the management of recurrent respiratory infections and those arising from the altered cardiovascular dynamics created by surgery as well as the primary defect. The good operative result in case 3 most likely depended on the presence of an adequate pre-existing right-to-left shunt at the level of the atria. It is interesting to speculate that in the presence of pneumonia there was a significant increase in pulmonary capillary pressures which were transmitted back through the shunt to the left ventricle, and then back across the atrial defect to account for the apparent increase in systemic venous pressures.

In case 2 the problem was more one of increased left ventricular workload resulting from the surgically caused left-to-right shunt. This, combined with the increased ventricular work from fever, led to high output failure. The delayed onset of mild failure is something which one would expect in any large series of postoperative tetralogies. One of the more common patterns described by Gross[1] was the onset of cardiac failure early in postoperative course resulting from the additional burden placed on the left ventricle. It was his experience that if there was no evidence of failure within five or six months, there would be little risk of this later on.

Other possible late complications, including a thrombosis or contracture at the site of the shunt and continuing susceptibility to subacute bacterial endocarditis, should also be kept in mind. Following the ligation of the anterior component of a double aortic arch, Gross noted marked improvement in the tracheal airway but there may be some persisting deformity in the tracheal cartilage, as evidenced by intermittent stridor in case 1. There appears to have been some resolution of the tracheal compression in this case, however, and there are fewer recurrent infections inasmuch as tracheal development is now proceeding along more physiologic lines.

None of the patients required oxygen for the episodes described above, although it was kept in mind for case 2. All were maintained on supplemental vitamins and were seen regularly in the outpatient pediatric clinic.

With regard to the choice of a digitalis preparation, digoxin has proved most convenient. For example, in case 2 a digitalizing dose of 0.066 mg per kilogram was used, corresponding to the dosage ranges used by Sapin, Donoso, and Blumenthal.[2] Their studies suggested the use of 0.075 mg per kilogram during the first year of life, with a maintenance dose of 33 per cent of the digitalizing dose. Slightly lower dosage ranges were used in the older age groups. Another advantage of digoxin is its reasonably rapid onset of action and the several forms in which it is available for clinical use. This includes a liquid preparation with which maintenance dosages can be more easily controlled in small infants.

In transitory heart failure associated with mild respiratory infection a satisfactory response may be obtained in some cases by the use of 0.025 ml of Mercuhydrin (brand of meralluride) per kilogram per day in lieu of digitalization. On the other hand, as illustrated by case 3, the use of cardiac drugs may not be necessary because the treatment of the respiratory infection may result in the rapid resolution of mild heart failure. Not needed in our group, but indicated in severe dyspnea, is morphine sulfate, 1.0 mg per 10 lb body weight.

Much of the cardiac symptomatology between acute episodes has been controlled by the spontaneous limitation of activity by the children themselves.

SUMMARY

In each of three cases of congenital cardiovascular malformations observed at this hospital there has been an excellent operative result. Some of the problems referable to a combination of the original defect and the altered cardiovascular dynamics which were manifested at various intervals following surgery have been described. The plan of treatment outlined includes early and vigorous therapy of respiratory infections, the judicious use of digitalis, and maintenance of adequate nutrition and general health. The increasing frequency of this type of pediatric patient and the excellent response to standard methods of treatment have been emphasized.

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CHILD GUIDANCE CLINIC: PRINCIPLES AND CRITERIA FOR ESTABLISHMENT AND EVALUATION

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IN THE 10-year period since the establishment of the first child guidance clinic in a military hospital,[1,2] several similar type units have been organized not only in the United States but also in overseas' commands. The intent, undoubtedly, has been to meet effectively the many needs and demands for the specialized services usually provided in a clinic so designated. In terms of the over-all mission of the medical services of the Armed Forces there is full justification for the maintenance of child (parent) guidance units.

However, in efforts to serve military parents who have children with problems, or whose unusual adjustment contributes to anxiety in the parents, some practices may well be employed which could do more harm than good. It is an application of the principle "A little knowledge may be a dangerous thing." A recent article[3] in the *U. S. Armed Forces Medical Journal* serves to underscore the need for caution and sound professional knowledge in setting up a child guidance program, full or "modified."

A working hypothesis that "professional services offered should meet highest possible standards in practice or not be available" should be the guide to any who plan, or have already organized, a child guidance clinic in a military medical installation. Sufficient data and professional opinion in civilian and military settings have been accumulated to serve as guideposts for those interested in extending specialized assistance to parents and children.

It is believed that a presentation of basic working principles and proposed criteria for the establishment and maintenance of a child guidance program in military medical units is needed and can be constructively useful. Prior to listing suggested criteria, a few pragmatic principles are presented for consideration.

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PRAGMATIC PRINCIPLES

The *need* for child guidance services exists whenever there are more than a few hundred military families for whom medical service is provided on an outpatient basis. Any practicing pediatrician or psychiatrist can supply ready-documented opinion on this point.

Once established, a clinic has such a heavy demand for services that a backlog soon develops. The largest and "best" clinics (civilian as well as military) cannot possibly meet all requests for treatment. Therefore, a reality principle requires acceptance of this fact and that measures should be taken to control the situation. This can be done by recognizing that *first* priority in time demand must go to active duty military patient needs. Therefore, blocking out a child guidance clinic schedule (adapted to local conditions) and holding to it is a necessity. Deviation from this, except to deal with a unique situation, will only result in a scattering of effort and time taken from other military requirements.

A clinic of this type requires a multi-discipline team working in close cooperation. The pediatrician, psychiatrist, neurologist, social worker, and clinical psychologist are key personnel who are needed inasmuch as any situation brought to the clinic involves the family unit (not the child alone).* Therefore, a basic principle may be stated that effective liaison and communication must be established. This can be done most efficiently through inclusion of such specialists as are needed on the clinic roster (regardless of primary duty designation). Staff personnel of other services for children should be invited and encouraged to participate in case conference sessions, *e. g.*, in a general hospital, physical medicine, dermatology, and ENT. Frequently, psychosomatic and primarily organic symptoms as occur in cerebral palsy, and postpolio conditions, present problems of a mental hygiene nature, where guidance may assist in obviating or relieving emotional problems.

Inasmuch as a child's primary occupation is that of a pupil, it is essential that effective communication be maintained with the schools in the area. A guiding concept of a well operated clinic includes this action as a standing operating procedure. In view of his training, this liaison function is best delegated to the staff social worker. As in any good communication, there is a mutual exchange of pertinent information concerning the welfare of the child, but without medical personnel violating personal family data, which should be kept strictly confidential.

Another principle, too often not observed, is that the referring physician should receive a report (if on the local staff, he should be invited to participate in the professional staff diagnostic

*Traditionally, the operation of a child guidance clinic has been a responsibility delegated to the Chief Psychiatrist.

discussion) of the clinic appraisal of the family situation. If treatment is indicated, he should be given the opportunity to continue with the treatment or request the clinic (or other unit) to carry on. Too frequently, a patient referred for consultation becomes the "property of the clinic," without the concurrence of the doctor who initiated the consultation referral.

Another principle is that the child guidance clinic should not provide services needed by patients that are available to them elsewhere. For example, if a school in which a patient is enrolled provides speech correction or remedial reading, or both, the clinic should not take over this function, although the staff may be equipped to offer these services.

Medical records on patients examined or treated in the clinic, should be kept on file for at least six years and not disposed of as are other records. The regulations on convenience files and for "research and training purposes" permits this to be done. Because of the nature of the problems usually treated in such a clinic, there is often need for future reference to these charts.*

It is vital that there be recognized the concept that there are no short cuts in the treatment of problem situations seen in a child guidance clinic. Scanning examinations, words of "advice," simplification of diagnostic study because of "expediency demands," "sending parents home to carry out suggestions," et cetera, are not present in a competently operated clinic.

Subsequent to direct diagnostic evaluation and staff discussion, resulting in at least tentative diagnostic and prognostic opinions, it is essential that the parents be seen for at least one interpretive type session, even if it is to be the terminal one—*e. g.*, in the event that treatment by the clinic is not feasible or possible, or, where treatment is not indicated. The operating procedure should ensure that this is done by the designated staff member. The same person should be certain to forward a clinic report to the referring physician.

All children seen in a child guidance clinic should have a physical examination, preferably by a pediatrician. The findings should be available to the psychiatrist when the child is first seen. As a working principle, it is desirable to request parents to have this done if the case was not referred initially by a pediatrician when informing them of the date of the initial clinic appointment. The parent can be requested to have such a physical examination done in the medical unit which serves the family's location, and to ask the physician to forward a copy of his findings directly to the clinic. The child's health record also should be brought in (as to any other clinic). Observance of this principle

*For example, in 1955 while in Germany, in a most difficult adjustment problem case, the author sent to Brooke Army Hospital for the case folder on the patient treated there in 1949. He received it promptly and it served its purpose very effectively.

is not only an economy measure but prevents overlooking essential medical data.

When a parent requests the clinic's service on the advice of the school principal or guidance counselor, the parent can be requested (in initial conversation) to ask the school principal to forward a brief report to the clinic. It has been found to be most practicable to be specific in requesting such reports. As a minimum, the following should be included: (a) scores on any standard tests administered to the child; (b) teacher rating of the child on such items as acceptance by classmates, special interests and abilities, special disabilities or handicaps, social adjustment in school, level of academic work in terms of grade expectancy; and (c) any data pertinent as to why the child was recommended to be seen at the clinic. A mimeographed sheet of paper with the aforenoted outline can be given the parent to assist in the accurate transmittal of the request (address of the clinic should be included).

It is an important working concept that both parents be seen together at the clinic, at least in the initial history phase of the evaluative procedure. Of course, if the father is away on extended TDY, or at an overseas station, this is not possible. Because it is a family matter, it is only proper that both parents be treated with equal respect; and also to determine relative degrees of awareness of a "problem." Certainly, such a session reveals much to the examiner concerning relative parental roles and intercommunication.

Staff conferences should be scheduled as regularly as are the clinic appointments. It is absolutely essential that the various examination findings and impressions be pooled and mutually evaluated to arrive at a definitive, albeit tentative, diagnostic opinion plus an estimate as to the need for treatment, if any. In a well run child guidance clinic this many-discipline coordination around one table provides not only excellent cross-training for personnel participating, but a more sound professional opinion than one made unilaterally. As necessary as is such a staff session for initial evaluation, so is also a staff review after therapy has been under way for a few weeks or months. In any hospital which provides residency training in psychiatry, neurology, and pediatrics, as well as internship training in clinical psychology and field work in social work, the establishment of such a clinic is essential to provide proper training to meet diplomate requirements.

The foregoing principles epitomize some key points of a practical nature. They are not all-inclusive but serve to delineate the responsibilities involved when specialized mental health services are offered to patients.

With this preface on principles, the following proposed criteria for a child guidance clinic are presented. They could be used by a visiting officer as a guide.

CRITERIA

1. The designated clinic should include on its basic staff at least one qualified psychiatrist, a social worker, and a clinical psychologist. Each of these should have prior training and experience in child guidance procedures.

2. There should be available in the same medical unit (or in one close by) a qualified pediatrician and neurologist. It is desirable that other medical personnel be present in the hospital, to whom referral may be made for specialized evaluation when needed.

3. The clinic should be established in a setting where military patients are not seen at the same time. (For example, a mental hygiene clinic located in a troop training area is not desirable. Instead, the clinic should be held in the hospital or post dispensary area.)

4. The clinic should have a room set up as a play diagnostic and therapy room, with proper equipment available for diagnostic and therapeutic purposes.

5. The waiting room should be equipped with some items geared to children's level. These can be removed on the days when the child guidance clinic is not in session.

6. The clinic should be operated at a designated time, and patients seen only on an appointment basis. In a military setting, such a clinic should have a minimum of four hours, and a maximum of eight hours weekly, for patient appointment time.

7. The clinic should schedule a period of at least two hours weekly for case conference discussion purposes.

8. The case conference discussion group should be composed of assigned personnel (who see patients in the child guidance clinic) as well as neurologists and pediatricians. Residents and interns should be invited to sit in on such conferences.

9. The scheduling of "new" patients should be done only for one half of the clinic appointment time. The other half should be devoted to treatment time.

10. When a child is in therapy the parents (or one parent, in an unusual case) are treated concurrently by another staff member.

11. If a child patient under treatment is enrolled in a school, liaison should be maintained with the proper school personnel when this is indicated (in most instances it is).

12. An effective record book (log book) plus a numerical file and record system should be maintained at all times, separate from other (*e. g.*, adult outpatient) clinic records.

13. Treatment-note records should be kept current by the personnel who treat patients in the clinic. Any staff conference

notes, correspondence, notes on forwarding of reports, et cetera, should be kept in the patient's folder. The file should be maintained in a location readily accessible to the staff.

14. If there is a resident in pediatrics on the hospital staff, he should be assigned to the clinic for a three- to six-month period as a staff member. Second- or third-year residents in psychiatry should be assigned a minimum of a total of six months during their last two years. If a clinical psychology intern is assigned to the hospital, he should be on duty in the clinic for a three-month period.

15. Effective liaison should be maintained with all other clinics in the hospital which include treatment of children in their specialties.

16. Provision should be made for proper supervision of personnel assigned to the clinic in training status.

17. Treatment should be conducted by various staff personnel within the range of their known special skills, as determined by the chief of the clinic.

18. There should be good follow-up on patients who had been in therapy but were temporarily terminated (to be "on their own" for a trial period).

19. Group therapy for patients should be conducted whenever feasible. It should be supplemented (when necessary) by individual treatment.

20. Scheduling should be done efficiently and economically to prevent unnecessary trips to the clinic, especially during the diagnostic period. (*For example:* The history should be completed one week, without the child being brought in; the parents and the child are then scheduled to come to the clinic during another week. The history and other data are made available to the examiners prior to their seeing the patients. If the psychologist is to examine the child, he will be scheduled to do this while the parents are seen by the psychiatrist; the child may then be seen by the psychiatrist.)

21. In most instances of diagnostic study the clinical psychologist will examine the child the day that the psychiatrist interviews the parents. Thus, at the staff conference there will be available medical history data and the current physical examination report, plus reports by the social worker, psychiatrist, and psychologist. Also the key family members will have been appraised by *at least two* staff members.

22. Expert consultants in child psychiatry and psychology should be available to the staff for teaching and consultation purposes (desirable but not mandatory).

SUMMARY

The principles for child (and parent) guidance services in military medical installations include the establishment of (1) local need for such specialized services, (2) an awareness that the services do not duplicate those otherwise available, (3) organizational structure of this type of clinic, (4) basic staffing requirements, and (5) essential liaison functions. There is need for long-range record keeping, functionally effective interdiscipline working relationships, and operating procedures that include attention to other members of a "patient's" family. Criteria for evaluation of a child guidance clinic are presented as guidelines both for establishing a clinic and for appraising one in operation.

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OFFICIAL RECOGNITION OF MILITARY MEDICINE

"I believe military medicine should be developed as a recognized specialty and given all the prestige and importance that can be given to it. At the same time the recognition of specialization as it is presently understood should be furthered. One of the fine things since the last war has been the program for the training of specialists within the services and the increased opportunity given to specialists to function in the framework of military medicine. I would advocate the organization of military medical men as a component part of the American Medical Association with status similar to a state medical society and with duly and democratically elected delegates to the house of delegates proportional to the number of doctors in the military services."

—RUSSEL V. LEE, M. D.
in *Journal of the American Medical Association*
p. 807, Oct. 19, 1957

SERVICE ARTICLE

MARTIN ARMY HOSPITAL, FORT BENNING, GEORGIA

Dedicatory Address

JAMES P. COONEY, *Major General, MC, USA*

SINCE the audience to which I am speaking today is chiefly composed of Fort Benning personnel and friends, I shall spend little time on the history of what is often called, and with good reason, the Army's most complete post.

THE PAST OF FORT BENNING

Fort Benning originated as Camp Benning, on a few acres near Columbus, Georgia, in 1918. It became a permanent military establishment in 1922, and by 1939, when its real functional expansion began, it had spread over almost 100,000 acres. During World War II it was the site of feverish military activity. Its expanded terrain of well over 200,000 acres made it, as it still makes it, admirably suited for all kinds of training. Infantry, airborne infantry, artillery, and armored units all trained here. Paratroopers learned both to jump and to fight. Young infantry officer candidates were graduated by the thousands. Army Specialized Training Program trainees were sent here. There was even a prison camp for Italian prisoners of war. The alumni of Fort Benning went to the far corners of the earth during World War II, they fought in the Korean Conflict, and they are still serving their country in all parts of the world today.

In the early days of what was then Camp Benning, its personnel was served by a station hospital which was housed in one-story temporary wooden structures. In 1924, a permanent hospital was completed. With two subsequent expansions, the original 105 beds were increased to 364. These hospital facilities were ample until 1939, when the prewar mobilization began. Then, as the military population increased to 75,000 and then to 100,000, these facilities also were increased, at first by improvisation, then by new construction, until the bed capacity was eventually more than 3,000.

Address delivered on 1 July 1958 at the ceremony of dedication. General Cooney is Deputy Surgeon General, Department of the Army.

THE NEW MARTIN ARMY HOSPITAL

The beautiful Martin Army Hospital which we are dedicating today is a far cry from the outmoded hospital of 1924 and even from the new structures of World War II. In its fabric and equipment it represents all that is newest and most efficient in hospital construction, including, as I am sure all of us gratefully acknowledge on this hot July day, its air-conditioning system.

The 500 beds with which this hospital will begin to operate tomorrow can be expanded to 1,000 beds. This is a wise provision. Last year, the hospital facilities which are now being superseded cared for total admissions of 11,604, representing an average daily census of 478 and 179,135 stay days. There were 338,172 visits to the outpatient clinics, and the clinical laboratories performed the incredible total of 540,743 tests.

The new Martin Army Hospital, Fort Benning, Georgia

I mention these statistics deliberately. This hospital has been designated to train interns and prespecialty residents, and the clinical material, as you can see, is ample for this mission.

MAJOR GENERAL JOSEPH I. MARTIN

For a great many years it has been the pleasant and well-deserved custom to name Army hospitals in the United States for great men in the Medical Corps and in other branches of the Army Medical Service. Major General Joseph I. Martin, in whose name it is my honor and privilege to participate in the dedication of this hospital today, was my friend, as I know he was the friend of many of you here. Let me recall to you some things about his long career in the Medical Corps and about the man himself.

I might begin with his remarkable ability to talk extemporaneously, yet so lucidly and so interestingly that few realized that his remarks, which were sometimes quite extensive, were entirely

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unprepared. Since I do not possess his ability, I will proceed with what a former Lord Chief Justice of England was in the habit of describing as his "carefully prepared impromptus."

General Martin never knew a medical life of which the Army was not a part. He was commissioned as a first lieutenant in the Medical Corps of the Organized Reserve immediately after he was graduated from the Chicago Hospital College of Medicine in 1918. Two years later, he was commissioned in the Regular Army Medical Corps.

Major General Joseph I. Martin

His first assignments were those of any young medical officer after World War I; not too many of them, I imagine, very exciting. But they were well performed, and, significantly, Captain Martin soon began to receive assignments for additional training. In 1926, he was graduated from the basic course at the Army Medical School at Carlisle Barracks. In 1928, he was graduated from the Advanced Course at the Infantry School here at Fort Benning. In June 1934, as a major, he was graduated from the two-year course at the Command and General Staff School at Fort Leavenworth, Kansas, and in June 1940, as a lieutenant colonel, he was graduated from the Army War College in Washington.

In the introduction to the story entitled "The Courageous Medics of Anzio,"* published in *Military Medicine* this year, General Martin, in the foreword, which he prepared shortly before his death, said that the medical officers who commanded there had developed their ability for their command as the result of long individual preparation and training. He might well have been writing of himself. All of his prewar assignments, a large number of which concerned medical education and training, prepared him for the responsible duties he was to assume during and after the war.

Those duties were impressive indeed: Surgeon, Fifth U. S. Army, in the North African (later Mediterranean) Theater of Operations; Chief Surgeon, Western Pacific, and, later, Surgeon, Army Forces in the Pacific; Commandant, Medical Field Service School, and Commanding General, Brooke Army Medical Center; and Chief Surgeon, United States Army Forces, Europe.

At Carlisle Barracks, when he attended the Army Medical School, General Martin was awarded the Skinner Gold Medal, which is presented annually to the student officer who is graduated with the highest scholastic average. It was the beginning of a long list of honors and decorations, including the Distinguished Service Medal; the Legion of Merit; the United States of America Typhus Commission Medal; and decorations from seven foreign countries, among them the Most Excellent Order of the British Empire.

The citations are remarkably interesting. The Typhus Medal, for instance, was awarded to General Martin for his activities in the epidemic in Italy in 1943-1944, during which no American soldier contracted the disease, and the epidemic in Japan in 1946, in which he had much to do with preventing what could have been one of the world's greatest medical catastrophes. The citation for the Legion of Merit recalled his co-ordination of the services of the British and American components of the Fifth Army, which also was recalled in the citation for the British order. The happy relations between the two medical forces, wrote the Commander of the Fifteenth Army Group, General Sir Harold R. Alexander, was due to General Martin's unfailing courtesy and consideration.

Similarly, I have found it extraordinarily interesting to follow General Martin's efficiency reports across the years. One of the earlier ones, which concerned his proficiency in horsemanship, carries the echoes of another day.

More seriously, he was described in the first report, in 1920, as a young officer of great good sense; he was then at Camp Grant, where, 20 years later, he was to do outstanding work in

*Bauchspies, R. L.: Courageous medics of Anzio. *Mil. Med.* 122: 53-65, Jan. 1958.

the organization and functioning of the Medical Replacement Center. Always the emphasis is the same: This was an officer who put as much energy and interest into distasteful work as into work in which his particular interests lay. He had a marked aptitude for field work and training. He would be well fitted for the training and command of medical department units for field service. He was of very great present and potential value to the Army. He would be the right hand of any officer under whom he might serve.

Then came the war and the postwar years when, in positions of high honor, trust, and enormous responsibility, General Martin's efficiency reports contain tribute after tribute, not only from the Medical Department officers who wrote them but from the line officers who countersigned them. Here are some of them:

> He combined splendid soldierly qualities and qualities of leadership with excellent judgment. He was professionally competent, and he was an outstanding authority on training and on the broad aspects of medical service in an overseas command. He was a superior administrator. He worked smoothly under adverse circumstances. *(The line officer who wrote that had seen him in action at Anzio.)* He was a creative thinker. He inspired respect, admiration and loyalty. He was of inestimable value to the service and superbly qualified for any assignment.

I imagine that the efficiency rating General Martin probably cherished most of all was the one that described him as a blunt, sincere, direct, demanding officer, who maintained a superior level of efficiency in his command. I think he must have cherished it because it was written by a line officer who had much the same qualities himself. This officer was General Anthony C. McAuliffe, Commander in Chief, U. S. Army Forces, Europe, the same General McAuliffe who some 10 years before had greeted the arrogant German demand for the surrender of the surrounded forces in Bastogne with the all-inclusive, absolutely final, and typically American response, "Nuts!" It is about the reply General Martin would have given had he been there.

Those of you who knew General Martin will remember him well. He was a big man physically, with a commanding presence and a booming voice. He took physical fitness and the technics of accomplishing it as part of the day's work. He could take poor physical material and make something out of it. As another of his efficiency reports noted, he had done just that with basketball and football teams of enlisted men when he was a young instructor at Carlisle Barracks.

Because of his broad service and experience and his assignments as student, instructor, and, later, commanding general at the various service schools, General Martin had hundreds of friends in the line. They admired and respected him, and they

listened to what he had to say because they knew that his presentations were authoritative and were based on a complete knowledge of his field. He would have been a fine line officer himself. As it was, he added to the prestige of the Medical Department and increased its capabilities for service because of his relationship to officers of the line.

He was known to thousands of Medical Department officers, noncommissioned officers, and enlisted men, who regarded service under him as a privilege, though there was no more demanding officer in the Medical Corps. One reason was that this same demanding officer had another, gentler, side. He was a man of deep religious convictions, who practiced his religion devoutly. He required the utmost of his men, but he had a deep understanding of human limitations and frailties, and no man who was doing the best of which he was capable need fear General Martin's frown. Many one-time sergeants who had become commissioned officers during World War II furnished problems in assignment after the war, but not to General Martin. He accepted them in his command, and he utilized these fine old soldiers to the best of their abilities and to the good of the service.

THE IDEALS OF AN ARMY HOSPITAL

What would General Martin have wished of this hospital which bears his name and which serves, among other establishments on the post, the school in which he was once a student officer? He would demand professional competence, of course. But I think he would also desire what a former President of the American College of Surgeons once well termed "the humanities of medicine."

As I have gone about from hospital to hospital in our worldwide Army Medical Service, I have been impressed again and again with the need for the practice of those humanities. We in the medical service must treat our patients as well as the diseases from which our patients suffer. We must approach their ills with kindly and sympathetic interest. We must remember, as Lord Moynihan once put it, that what to the physician is part of his daily life, to the patient and his loved ones may be the ordeal of a lifetime. If we bear these things always in mind, no detail of the care of a patient will be too small to be of concern to us.

This is the art of medicine. It should be practiced in all diseases, be they trivial or lethal. It holds for all who have any relationship at all with the patient and his family, be they medical officers, nurses, enlisted men, or civilian personnel. The art of medicine can be practiced, you know—and should be practiced—by the enlisted man with whom most patients in military hospitals have their first hospital contacts. It should be practiced by the dietitian, the physical therapist, the registrar, and the ambulance

driver, to mention a few of our ancillary personnel. Some of the most gratifying comments we are receiving about medical care concern the kindness and thoughtfulness of these personnel, and I hope that in this hospital they will always realize their responsibility for maintaining the honor of the Medical Corps. The art of medicine is made up, as you see, of little as well as big things.

Are the art and science of medicine incompatible? Not at all. Let me give you one or two illustrations:

Take William Beaumont, whose faithful performance of what he called "the common avocations of life and professional duties" prepared him so admirably to make the observations on gastric physiology which are the basis of all modern concepts in that field and which were possible when opportunity came his way in the person of "old fistulous Alex."

Take Sir James Mackenzie. His title came late. He was for most of his life a country doctor, with little medical education and with no laboratory personnel or equipment to help him, but no man has ever contributed more to the science of cardiology. He accomplished it simply by observation of his own patients over years and years and years, and by the careful recording and correlation of these observations. To the end of his days, his biographer recalled, he wore the title of family doctor "as if it were a plume."

Take Harvey Cushing, the pioneer and founder of modern neurosurgery. His biographer, the distinguished physiologist, John F. Fulton, wrote of him:

> By valor and divine aid he had more than fulfilled the promise and ambition of his youth: scientist, pathfinder, artist, writer, and bibliophile—yes, but above all, "a good doctor."

A recent President of the American College of Surgeons wrote his Presidential Address around that theme, "But above all, 'a good doctor.' "

Finally, take Sir William Osler. His academic education and medical training were, of course, superior. Before his life ended, there was scarcely a disease to which he had not added some illuminating observation. Yet one thing that he always told his students and residents was that they must have regard for the "rights" of patients, by which, of course, he meant far more than the legalistic implications of the term. To him, the rights of patients included the human kindness, the sympathy, and the compassion which I plead with you to make the hallmarks of this new hospital. In this scientific age they are sometimes forgotten. Osler never forgot. It was said of him that he never left a sickroom without leaving renewed hope behind him. He was the living embodiment of something Stephen Paget once said, that to

be wanted by men and women, to come natural to them in time of trouble, might fairly be called a career.

Among Osler's letters is one written early in his practice to the father of a young man who had died of smallpox in Montreal. He told all the particulars of the son's illness, and we may be certain that the patient had from the young physician the best care that medical science could then provide. But Osler also told the bereft father how he had read to him from the Bible the young man's mother had marked for him, and, he concluded, "I performed the last office of Christian friendliness I could and read the commendatory prayers at his departure." How it must have comforted those stricken parents to know that their dying son, a stranger in a far land, had gone into the farther country with his hand in the hand of this gentle and kindly physician.

Osler never lost that kindliness. Many years later, when he was Sir William Osler and Regius Professor of Medicine at Oxford, shortly before his own beloved and only son fell in battle, he wrote another grieving mother of how, the night before he died, her young soldier son had asked to be alone with him, and, he said, "I did what I could to comfort him." No physician ever lived in whom the science and the art of medicine were more happily combined.

It was in 1899 that Letterman General Hospital in San Francisco became the first Army hospital to bear the name of a former medical officer. The hospital which we are dedicating today becomes the sixty-fifth to bear the name of some officer in the Regular or Reserve Army Medical Service or some related area who, as the memorialization criteria express it, "has distinguished himself by serving in or with" the branch in which the installation has been set up.

I knew General Martin personally. I have also known some of these other officers during my service in the Medical Corps. Some of them I know only from their records. But one thing I know about them all: They have all of them done well by their country. And as I recollect the hospitals which bear their names—I have had some personal contact with almost all of them—I realize that these hospitals and their personnel also have done well by the country.

This hospital, beautiful, new, and modern in every respect, will, I know, continue to build upon the honorable traditions of the medical service that has been provided here since Fort Benning was first established 40 years ago. I can wish for this new hospital nothing better than that it should carry on in its own tradition and in the tradition of the other hospitals which, like it, bear the honored names of former medical officers. I know that the gallant and distinguished officer whose name it will bear could wish nothing better for it. And I know that he would wish for it, as I do, that its record of human kindness should be as great as I am sure its record of scientific achievement will be.

Clinicopathologic Conference

U. S. Naval Hospital, St. Albans, N. Y.*

FEVER AND HEPATOMEGALY

Summary of Clinical History. A 55-year-old retired Army officer noticed two abdominal masses two years prior to his first hospital admission. One mass was on the left side of the abdomen, the other in the epigastrium; both had gradually increased in size and were occasionally tender. The patient was known to have had (1) pleurisy on the right side in childhood; (2) an appendectomy in 1925; (3) a tonsillectomy in 1940; (4) malaria in 1944 and 1946, treated with Atabrine (brand of quinacrine hydrochloride); (5) fracture of the lumbosacral spine with disk herniation in World War II; (6) rectal polyps removed by sigmoidoscopy in 1946; and (7) a fibroid tumor removed from the right forearm a few weeks prior to admission. He was a lawyer, and had served in Africa and Korea.

FIRST ADMISSION

On 11 August 1955 the patient was admitted to this hospital with chief complaints of abdominal pain, weakness, and melena of one-week duration, plus the known abdominal masses. His pain was aching, most marked in the right upper quadrant and epigastrium, not related to food ingestion (except for relief following milk), and accompanied by anorexia. Although the patient denied alcoholism, his family told of frequent ingestion of alcohol.

Physical Examination. The patient was a well-developed, well-nourished man who appeared chronically ill and anemic, but in no acute distress. His temperature was 99.4°F; pulse, 108; blood pressure, 180/104 mm Hg; and respirations, 20 per minute.

*Capt. Herman A. Gross, MC, USN, Commanding Officer. From the Laboratory Service, Capt. Sarkis S. Sarkisian, MC, USN, Chief.

The abdomen was flat and soft, with a mass thought to be spleen in the left upper quadrant, and an epigastric mass thought to be liver. Both masses were tender. The prostate was minimally enlarged. There was tenderness over the lumbosacral spine with straightening, and rigidity of the lumbar curve. The remainder of the physical examination was negative.

Laboratory Studies. Examination of the blood revealed a red blood cell count of 2,330,000 per μl, a hemoglobin of 6 grams per 100 ml, and a hematocrit of 22 ml per 100 ml. Sedimentation rate was 30 mm per hour (corrected). The white blood cell count was 6,050 per μl with 80 per cent neutrophils, 1 per cent band forms, 17 per cent lymphocytes, 1 per cent monocytes, and 1 per cent eosinophils. The platelet count was 123,490 per μl. Bilirubin was 0.5 mg per 100 ml; total proteins, 6.0 grams (3.0 grams albumin, and 3.0 grams globulin) per 100 ml. Sulfobromophthalein sodium retention was 15 per cent in one hour. Serum amylase was 416 units; thymol turbidity, 5.9 units; alkaline phosphatase 12.1 King-Armstrong units; and the cephalin-cholesterol flocculation test was negative. A Coombs test was negative.

A roentgenogram of the chest disclosed no abnormalities. A gastrointestinal series revealed an enlarged spleen which displaced the stomach to the right and the right kidney medially and downward. An extrinsic pressure defect was demonstrated on both the greater curvature of the stomach and the duodenal cap. A cobblestone appearance of the cardia suggested varicosities. An esophogram showed large varicosities extending from the lower half of the esophagus to the level of the tracheal bifurcation. An intravenous pyelogram was normal. Films of the lumbosacral spine were consistent with hypertrophic arthritis.

Course in Hospital. Repeated blood transfusions raised the red blood cell indexes to normal. Liver biopsy showed portal cirrhosis; bone marrow examination was normal. Rectum-to-arm ciculation time, using sodium^{24}, was 90 seconds (consistent with increased portal vein pressure). Two months after admission, a splenorenal shunt was performed, with concomitant splenectomy. At operation, the liver was described as small and nodular. Initial omental venous pressure was 360 mm of water, and, after completion of the shunt, the pressure fell to 300 mm of water. The patient tolerated the procedure well, and postoperatively his anemia did not return. However, he developed a low-grade fever, which was present on discharge. Two months later, on return outpatient visit, his rectum-to-arm circulation time was 33 seconds (consistent with a patent shunt).

SECOND ADMISSION

The patient was readmitted on 10 June 1956, complaining of fever and left upper quadrant pain. Since discharge he had experienced continuous fever with spikes up to 104°F.

Physical Examination. The patient was alert, co-operative, and did not appear to be in acute distress. His temperature was 100.2°F; pulse, 88; blood pressure, 148/56 mm Hg; and respirations were 16 per minute. Large, soft submandibular lymph nodes were noted bilaterally. The abdomen was soft. There was a curved linear scar on the right flank, and a "hockey-stick" scar on the left flank and back. There was mild tenderness in the left upper quadrant. The liver was firm, and was palpable four fingersbreadth below the right costal margin. The flanks appeared full, but without ascites. The lumbosacral spine was tender. Physical examination was otherwise negative.

Laboratory Studies. Complete blood cell counts were within normal limits. Urine sediment contained from 2 to 4 white and 2 to 4 red blood cells per high-power field. Serum albumin was 3.6 and serum globulin was 4.6 grams per 100 ml. Alkaline phosphatase was 12 and acid phosphatase was 2.5 King-Armstrong units. Thymol turbidity was 10.2 units. Cephalin-cholesterol flocculation, febrile agglutinins, and repeated blood cultures were negative. Neither ova nor parasites were found in many stool specimens. Malaria parasites were absent from smears. Bone marrow examination was normal.

Roentgenograms of the chest and abdomen revealed no abnormalities; those of the gastrointestinal tract showed progressively enlarging varices of the esophagus. An intravenous pyelogram disclosed no abnormalities.

Course in Hospital. The patient gradually developed ascites and anemia. He was given a course of emetine hydrochloride and chloroquine phosphate in spite of the absence of demonstrable parasitic infestation, but his febrile course was unaltered. On 15 August, two months after his second admission, exploratory laparotomy was performed. At exploration, the shunt was believed to be patent. Pressure in the omental vein was 300 mm of water. The patient tolerated exploration well, but continued febrile with occasional spikes to 104°F. On 25 September he had massive hematemesis, controlled by passage of a Blakemore tube and blood replacement. Nine days later he died following another massive hematemesis.

DISCUSSION

Doctor Hochman:* The problem presented by this patient is the explanation for the continuous fever that was present since the patient had an operation for portal hypertension. Two very enticing possibilities immediately arise, one from the past, and the other from the present history. The patient had malaria on two occasions; could this present pyrexia be due to a recurrence of malaria? Not very likely, for we are told that repeated examinations for malarial parasites were all negative. The course of the fever is not typical, for this was a prolonged, continuous fever over a 10-month period.

*Lt. Richard I. Hochman, MC, USN, Resident, Medical Service.

Thrombosis of the splenorenal shunt must be considered. There are several arguments against this hypothesis; first, the rectum-to-arm circulation time, considered to be a reliable test, was within normal limits; second, at exploratory laparotomy, the shunt was thought to be patent. In spite of these findings, thrombosis of the shunt must still be strongly considered. Enlargement of esophageal varices, development of ascites, left upper quadrant tenderness, and elevation of omental venous pressure to 300 mm water (normal is 60 to 80 mm water) are all in favor of thrombosis of the shunt.

From the protocol, we see that amebiasis was considered, as evidenced by the therapeutic test with emetine and chloroquine. Amebiasis should be suspected in every fever of unknown origin. Repeated examinations of fresh, warm stools should be made, to rule out amebic infestation. If negative, and the diagnosis is still strongly considered, sigmoidoscopic examination for amebic ulcers should be performed, and appropriate specimens obtained. Finally a therapeutic test, as was done here, should be tried.

The patient's military career brought him to Africa, and tropical disease must be considered. Schistosomiasis must be ruled out, and here many stools were negative for ova and parasites. *Schistosoma mansoni* and *Schistosoma japonicum* can both cause such a clinical picture, with the former more likely, because *S. japonicum* usually produces a more severe, acute disease, but may carry on to the chronic stage of hepatic cirrhosis. *S. mansoni* commonly produces a cirrhotic picture, with hematemesis from esophageal varices as the terminal event. The initial infection may be overlooked.

Infections such as tuberculosis, typhoid fever, and brucellosis must be considered in all unexplained fevers. We are told that gastric washings and agglutination tests were negative.

Lymphoma is another possibility in unexplained fevers. But, physical examination did not reveal any significant adenopathy. Blood cell counts and bone marrow examinations likewise were not suggestive. The fever was not of a Pel-Ebstein type, which, incidentally, is not commonly found in proven cases of Hodgkin's disease. Roentgenograms of the chest did not reveal any mediastinal widening, which may be the first sign of lymphoma. No intra-abdominal masses, aside from the enlarged liver, were palpated. Uric acid and serum calcium determinations would have been helpful, for hyperuricemia may result from increased catabolism while hypercalcemia may reflect bony involvement with tumor. Lacking substantiating evidence, however, lymphoma may be dismissed.

Other malignant processes might be considered. In this case, the possibility of hepatoma is interesting. Primary carcinoma of the liver occurs in about 8 per cent of all cases of cirrhosis. It commonly produces a persistent, irregular fever, which may be caused by necrosis of the tumor nodule. If, in a patient with cirrhosis, a careful search for other causes of fever is unproductive, then hepatoma must be put near the top of the list of causes. Liver biopsy may be helpful, if

the operator is so fortunate as to biopsy a malignant focus. Ascites in a patient with cirrhosis and without hypoalbuminemia is suggestive of hepatoma. A marked elevation of the alkaline phosphatase without accompanying jaudice is also suggestive of hepatoma. Ascitic fluid should be examined for malignant cells.

The cause of death in this patient was probably a result of the hematemesis. As for the continuous fever, I shall put hepatoma first and chronic schistosomiasis as the next best cause.

Doctor Le Blanc:* When this patient was first seen by physicians from the surgical service, he fulfilled the criteria of what would be considered a good candidate for a shunting procedure. The history of a heavy alcoholic intake together with the physical and laboratory findings, including a liver biopsy consistent with Laennec's cirrhosis, left little doubt as to the cause of his portal obstruction. The roentgenographic evidence of large varices of the lower esophagus and cardia of the stomach, together with the elevated rectum-to-arm circulation time, splenomegaly, and hypersplenism, reflected an elevated portal pressure. The cause of the obstruction, the resultant portal hypertension, and the evidence of bleeding from the varices were then manifest. With no ascites, a negative cephalin flocculation, a serum albumin of 3 grams, and only a moderately elevated sulfobromophthalein retention, the patient was considered a good operative risk for a shunting procedure. The decision remained then only as to choice of shunt. A markedly enlarged spleen and evidence of hypersplenism were the deciding factors in performing a splenorenal anastomosis. The removal of the spleen would relieve the symptoms of hypersplenism as well as decrease the portal blood volume by an estimated 40 per cent.

The fact that the omental venous pressure dropped only from 360 mm to 300 mm of water after the shunting was completed is not an unusual finding. The decrease in the size of the varices and the drop in portal venous pressure are gradual processes, and it takes an estimated several hours before the body accommodates to this shunting procedure. The normal rectum-to-arm circulation time two months postoperatively indicated to us that the shunt was patent at that time. The possibility of a hepatoma was considered by members of the surgical service but this was never confirmed by several deep-needle biopsies in various quadrants of the liver. This, however, did not negate our suspicions. The increased incidence of a hepatoma in a cirrhotic liver is well known.

In view of the patient's symptoms of left upper quadrant tenderness and febrile course, a limited abdominal exploration was performed to determine if an abscess was present. No infection was found at time of operation, and the liver was not palpated.

This case illustrates one of the major disadvantages of a splenorenal anastomosis. For reasons not well known, possibly due to the angle

*Lt. Gilbert A. Le Blanc, MC, USN, Resident, Surgical Service.

of the anastomosis or movement of the viscera, delayed closure of the anastomotic site does take place.

Clinical diagnoses:

1. Portal cirrhosis
2. Hepatoma
3. Esophageal varices with hemorrhage
4. Schistosomiasis (?)

PATHOLOGIC FINDINGS

Doctor Hyams:* The body was that of a well-developed, well-nourished man of recorded age. Externally, there were icteric scleras, as well as an icteric tint to the skin surface. The abdomen was protuberant with a definite fluid wave. The legs revealed a 1-plus pitting edema of both ankles and feet. The thoracic cage contained approximately 100 ml of blood-tinged fluid in both pleural cavities. The heart was normal on gross examination. The lungs contained some frothy red fluid in the dependent portions bilaterally. The abdominal cavity contained approximately 1,000 ml of clear amber-colored fluid. The spleen was absent. There was only a small, healed, linear scar with remnants of black silk sutures in the left renal vein. The splenic vein was not identified. The liver weighed 2,300 grams and was of normal shape. The external surface had a tan, markedly nodular appearance (fig. 1) with the exception of the inferior surface of the right lobe where there was a large, rounded, tan, soft protruding mass that was 12 cm in diameter. The cut surface of the liver revealed the typical tan hobnail appearance, except in the right lobe (fig. 2). The center of this lobe was replaced by a pinkish-white caseous mass, which appeared to be circumscribed by a thin band that resembled fibrous tissue. The stomach contained 1,500 ml of dark-red blood, and the intestine was filled with old blood. A ruptured varix was identified in the lower third of the esophagus. The remaining findings were normal. Microscopically, except in the area of the right lobe where there was mainly caseous necrotic material, the liver showed the typical picture of portal (Laennec's) cirrhosis. However, there was an occasional small island (fig. 3) composed of cells resembling hepatic cells but showing marked pleomorphism and hyperchromatism of the nuclei, as well as an indistinctness of the cytoplasmic borders and numerous mitotic figures. These cells were characteristic of a hepatocarcinoma (hepatoma). The remaining microscopic findings were pulmonary edema with evidence of aspiration of recent hemorrhage, esophageal varices, atrophy of testes, and chronic cystitis of the urinary bladder.

In recapitulating the case, the direct cause of death was massive gastrointestinal hemorrhage from a ruptured esophageal varix, a consequence of long-standing portal cirrhosis. The

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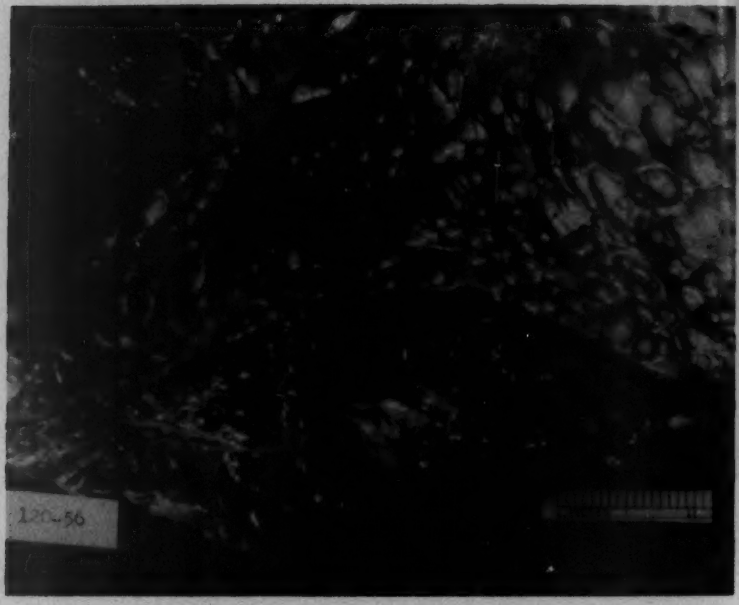

Figure 1. External surface of liver, showing hobnail appearance.

Figure 2. Cut surface of right lobe of liver, showing tumor.

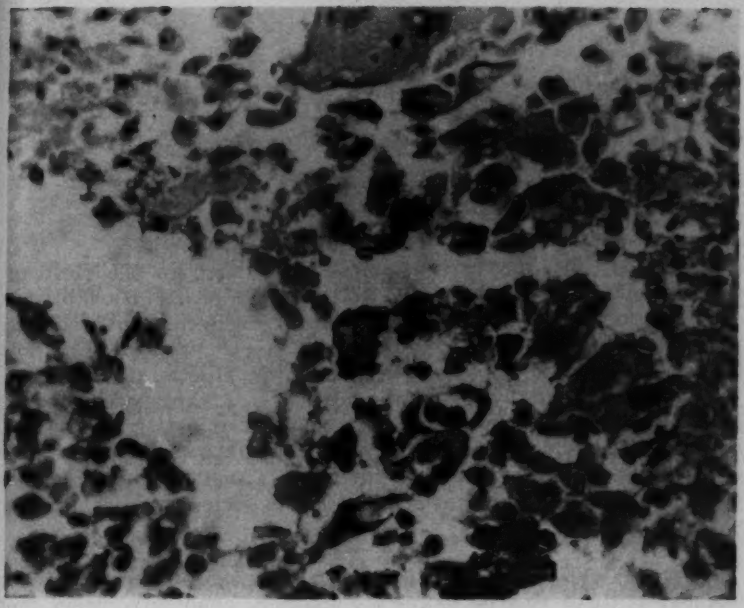

Figure 3. Photomicrograph of hepatoma. (× 200)

above course and termination is the usual picture in portal cirrhosis. The finding of hepatocarcinoma in cirrhosis of the liver is not unusual, and in the clinical picture of this patient, with an unexplained fever, hepatocarcinoma should be considered.

Pathologic diagnoses:

1. Hepatocarcinoma
2. Cirrhosis, portal
3. Pulmonary edema, bilateral, with aspiration of recent hemorrhage
4. Bleeding esophageal varices, with massive gastrointestinal hemorrhage

CASE REPORTS

Speculations Concerning the Influence of Psychoneurosis on Cardiac Pain

ROBERT SPENCER, *M. D.*

DURING the last two decades of research in the field of cardiovascular disease, more emphasis has been placed on correlation of phenomena observed by both physiologic and psychologic technics. Great progress has been made by improved methods to apply and evaluate the electrocardiogram on the one hand and by the daily advances in the exploration of psychiatry on the other. Though many links are still missing for an adequate understanding of the dynamic factors that may play a causative role in cardiovascular disease, more and more clues have emerged from this combined effort to focus research to both aspects.

The clinical diagnosis in cases of precordial pain, for instance, at times may be extremely difficult, especially when the patient also exhibits a variety of anxiety symptoms. The diagnosis will largely depend on the history and cannot be proved or disproved by the demonstration of psychoneurosis on the one hand or of organic heart disease on the other. It is well known that anxiety may have an adverse influence on myocardial ischemia while its resolution may be followed by at least temporary relief of precordial pain and such a case may have to be followed for a considerable length of time before a correct diagnosis can be made.

An unusual case of fatal cardiac pain overshadowed by psychogenic manifestations is reported.

CASE REPORT

A civilian employee had frequently been seen at the dispensary of this hospital since 1951 when he was 36 years old. He then complained of vague pain in the right side of his chest, shoulder, and neck. The pain responded well to courses of physiotherapy. During the next four years, *i. e.*, from 1951 to 1955, he frequently called at the dispensary for a variety of aches and pains in the right side of his chest, left maxilla, left big toe, et cetera, but examination always

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revealed a healthy, slightly overweight, but not actually obese, middle-aged man, who looked at least five years younger than his stated age.

On three occasions he had to be admitted to the hospital for observation, investigations, and reassurance. The patient always became asymptomatic the day after admission, and investigations, including complete blood cell counts, serologic tests for syphilis, blood uric acid, urinalysis, roentgenograms of the chest, cardioscopy, and repeated electrocardiograms with Master's two-step exercise test were all negative (fig. 1, A and C). On each occasion it had been noticed that the patient was very introspective and he appeared particularly concerned about his heart.

In 1955, at the age of 40, in the course of an annual physical examination which was negative, an electrocardiogram had been taken which revealed frequent ventricular extrasystoles in groups of three and four. There were otherwise no abnormal features in the cardiogram. The patient was not informed of these cardiographic findings, which at the time were considered to be of no clinical significance. Four weeks later, however, the patient came to the dispensary complaining of discomfort in the left side of his chest which was not described as an actual pain but had "bothered him off and on" for the last 24 hours.

Observation in the hospital during his second admission revealed a blood pressure of 170/110 mm Hg and a regular but fast pulse of 120 per minute on the day of admission. There was no evidence of premature beats and an electrocardiogram and examination of the heart were negative. Further blood pressure recordings varied between 120/90 to 145/95 mm Hg. The patient complained of heaviness in the left inframammary area, rather localized without radiation, and there was also a toothache-like pain in the region of the left maxilla. From time to time he would break out in a profuse sweat and he also admitted that he had become rather worried lately since the discomfort in his chest had now shifted to the left and was no longer "on the wrong side." By this he meant that he now associated his sensation with heart disease.

After his discharge from hospital he was again frequently seen at the dispensary complaining of excessive fatigue, frequent racing of his pulse, slight dyspnea on climbing stairs at home, and a peculiar and permanent feeling of inward tension and fear.

The findings on his third admission were as negative as on his previous ones. The blood pressure now remained quite steady, between 130/90 and 140/90 mm Hg, with a pulse in the region of 100 per minute and a regular rhythm. The patient, however, complained of a variety of frightening sensations such as a feeling of racing and slowing of his pulse, shortness of breath off and on, and a periodic flickering of brightness and darkness in both eyes "like the effects of a rotating ventilator." Expert ophthalmologic examination failed to reveal any

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pathologic lesion; vision, visual fields, and fundi were normal, and it was thought that his symptoms might have been due to arteriospasm of the retinal vessels. As on previous occasions all his symptoms subsided after a few days, and a week later he was reassured and discharged.

For over three months the patient felt well and did not call at the hospital. When his symptoms recurred he was advised to see either a psychiatrist or a cardiologist for a second opinion. He decided in favor of the cardiologist, but expressed a willingness to see a psychiatrist later if thought necessary. The consulting cardiologist reassured the patient that no organic cardiac disease was present and in his medical report stated that he believed that the patient's complaints were due to a vasovagal syndrome, though at the beginning of his illness he might have experienced attacks of paroxysmal tachycardia. For the next two weeks the patient was well and felt "on top of the world." "It had all been in my mind" and "Maybe I read too much about heart diseases" were now his frequent remarks. He disclosed that when a friend of his had died suddenly of a heart attack he began to read about heart diseases. After this short asymptomatic period he suddenly started to complain of frequent attacks of sharp precordial pain which came on whilst walking only a short distance, as well as at rest at home. It was the first time that he had described a definite pain and not only a discomfort. The pain started in the epigastrium, radiated upward to both sides of his chest, and responded to glyceryl trinitrate.

Ten days later, in March 1956, the patient had to be readmitted for the fourth time by ambulance after his office telephoned that he was in severe pain and unable to walk to the hospital, a distance of 200 yards. He looked pale with a slight malar flush, and was apparently in great pain. He clutched his chest with cold, clammy, cyanotic hands, and there was perspiration on his forehead. The pupils were widely dilated. His pulse rate fluctuated between 84 and 120, its rhythm was regular, heart sounds were normal, and there were no murmurs, pericardial rub, or abnormal accentuation. Respiration was normal, and there was no evidence of pulmonary congestion. Blood pressure was 135/85 mm Hg. The attack had started half an hour previously and had not responded to glyceryl trinitrate. The patient had at first thought that he had an attack of indigestion, but later the pain extended toward the lower end of the sternum, radiating toward his left shoulder and down his left arm. Morphine, 0.016 grams, was given intramuscularly, after which the patient fell asleep. Next morning, apart from a slight feeling of general weakness the patient felt and looked perfectly well and did not complain of any pain. An electrocardiogram, however, now revealed for the first time deep depression of the RS-T segment in limb and precordial leads, most marked in I, II, V_4, V_5, and V_6, and elevation of RS-T in aVR and V_1, suggesting diffuse myocardial ischemia. No Q wave was seen, and T waves were upright except in aVR (fig. 1, B and D). Twenty-four hours later a perfectly normal tracing was recorded and all RS-T deviations had subsided.

Figure 1. A and C show normal electrocardiogram taken on 15 October 1955. B and D show RS-T depression present on 14 March 1956, the day following a severe episode of precordial pain.

The patient was kept in bed and was given sedatives and glyceryl trinitrate with instructions to suck one tablet in case of recurrence of the pain. An oxygen cylinder and cardiograph were placed near his bedside. He admitted now that he had been living under considerable mental and physical strain for several years. He disclosed serious domestic and financial difficulties as well as friction at his work, the latter had even led to a court martial proceeding. He thought he had noticed that his superiors had become dissatisfied with his work, and from his story a good deal of repressed hostility, fear, and severe anxiety with even some psychotic paranoid features appeared to emerge. It was now thought that his anxiety state had reached such a degree that it had become responsible for attacks of coronary spasm and transient myocardial ischemia. The normal tracing 24 hours after the precordial pain had subsided seemed to confirm such a view. The patient, however, experienced more frequent attacks of precordial pain at rest in bed, which came on during the day and night. The pain lasted only a few minutes and responded to glyceryl trinintrate. The patient had now become so frightened of these attacks that he took glyceryl trinitrate whenever he felt that a pain was imminent and it therefore became impossible to take an electrocardiogram actually during an attack. Those tracings that were taken after one showed a degree of myocardial ischemia, though in none of them were the changes found to be as marked as in the first one after admission. The mere recording of his blood pressure or the auscultation of his heart seemed to bring on an attack, and this was usually associated with flushing of his face, frequent blinking of half closed eyelids, and a nervous fingering of the sternal area. One day when he had felt comparatively well he suggested bringing on an attack at will so that a tracing could be taken during the pain. He added that he had only to think of some of his worries in order to provoke precordial pain. Throughout this stay in the hospital his temperature, white blood cell count, and sedimentation rate had remained normal.

On the seventh day of this last admission, he woke up at 0300 hours, asked for the oxygen mask, and died within 3 minutes.

Post-Mortem Examination

Gross findings. The heart weighed 360 grams. The myocardium was dull red and moderately flabby. The epicardium was smooth and glistening. The myocardium of the left ventricle measured 14 mm in thickness, and that of the right ventricle 5 mm. The cavities of the heart were slightly dilated and contained clotted blood. There were no palpable areas of myocardial softening. The endocardium was smooth and glistening throughout. The intima of the coronary arteries showed a few yellow nonobstructive plaques with the exception of a single plaque near the beginning of the descending branch of the left coronary artery. This plaque bulged into the lumen of the coronary vessel to reduce its internal diameter one third to one half of normal. The aorta showed good elasticity with scattered intimal plaques, without calci-

fication or ulceration. A detailed examination of all organs in the thoracic and abdominal cavities, including the adrenal glands, failed to show any abnormality.

Anatomic diagnosis: Localized obstruction of a coronary artery.

Histologic findings. Sections taken through a segment of coronary artery revealed thickening of the subintimal layers of the vessel by a pale basophilic type of connective tissue (fig. 2). In some areas the matrix of the connective tissue showed a fibrillar or mucoid type of degeneration and in these areas nuclei were largely absent or pyknotic. There was no evidence of ulceration of the intima, thrombosis, inflammatory cell exudate, or hemorrhage within the wall of the vessel.

Figure 2. Intimal thickening of descending branch of left coronary artery. (hematoxylin and eosin stain, × 5.5)

Sections of heart muscle and sections, through the atrium and ventricle including a portion of one of the heart valves, revealed normal endocardium, muscle fibers, and nuclei. Striations were evident on many of the muscle fibers. There was no apparent increase in fibrous tissue between muscle fibers in this section. The heart muscle showed fat cells within the pericardium extending somewhat into the myocardium between muscle fibers. This represented a mild degree of lipomatosis. There was no evidence of fatty degeneration of muscle fibers.

Further sections revealed a vessel within the pericardium showing increase in fibrous-like tissue of the subintimal layers. Much of the media of the vessel showed a degenerative type of intercellular material rather than muscle fibers. There was no evidence of thrombosis, ulceration of the intima or hemorrhage within the wall of the vessel.

Within the myocardium beneath this vessel there was some increase in fibrous tissue between muscle fibers. There was no inflammatory cell exudate in this section (fig. 3). Sections of the aorta revealed normal intima. The media of the vessel was formed of elastic fibers in the usual amount and distribution.

Microscopic diagnosis: Mild unobstructive coronary atherosclerosis with a single plaque in descending branch of anterior coronary artery reducing its bore by one half.

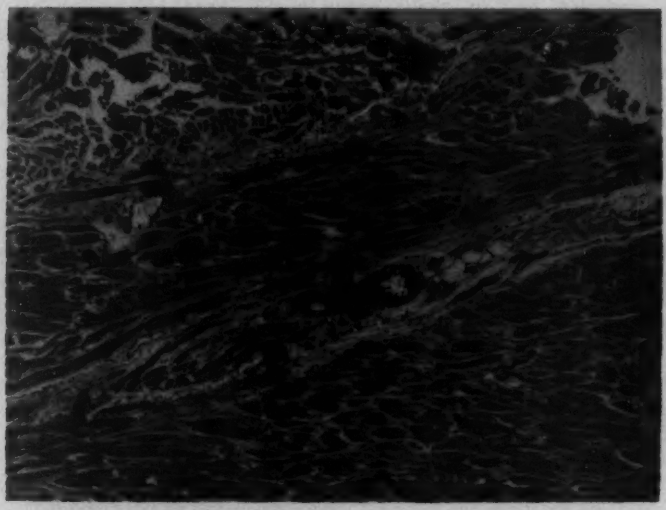

Figure 3. Area of left ventricle showing minimal fibrosis. (× 90)

DISCUSSION

The intimate relationship between the feeling of anxiety and sensations arising from the heart has long been emphasized by psychiatrists. These sensations may vary in intensity from slight precordial uneasiness, accompanying mild anxiety states, such as that experienced by a motorist when he is halted by a traffic policeman, to the intense "angor animi" that characterizes a severe attack of coronary thrombosis. It is said that John Hunter's terminal attack was due to anger, for he took exception to the fact that the appointments committee at one of the London teaching hospitals would not give a vacant position to a Scottish protégé of his. The fact that fear and/or anxiety frequently aggravates an already impaired coronary circulation is well known and universally accepted. Mere concentration on the heart may suffice to provoke a feeling of anxiety in predisposed individuals and is usually associated with an uneasiness under the sternum or left inframammary region. Whether intense and prolonged fear

can, in fact, lead to attacks of coronary insufficiency manifested by cardiac pain and demonstrated by RS-T segment changes in patients who were believed not to suffer from heart disease is still unproven, and indeed is unlikely.

It has become clear, however, that whatever the condition of the heart may be, the effect most closely associated with the heart is anxiety. A diseased cardiovascular system can produce anxiety just as anxiety is frequently a precipitating factor in cardiac symptoms; in other words, we are dealing with a typical vicious circle.

Klemperer[1] described emotion as an etiologic factor in organic heart disease and stated: "Modern medicine does not doubt that emotion can lead to cardiac death, but it has doubted whether joy and pain can paralyze a normal heart or whether a serious heart disease must not have been present before a strong effect can lead to sudden death." He asked whether a lasting emotion can so disturb a previously healthy heart as ultimately to threaten death, and after he cited a series of cases he answered this question in the affirmative. It was his opinion the finding of myocardial lesions in these cases is not against a psychogenic etiology and he explained them as the result of coronary spasm and resulting myocardial ischemia.

Friedberg[2] termed the wide range which exists between the coronary flow under basal conditions and the augmented flow, when requirements of the myocardium are maximal, "the coronary reserve." This reserve may diminish or even become exhausted, not only by physiopathologic processes but also through psychogenic causes. In fear and excitement the physiologic power to augment the coronary flow by reducing the resistance or by dilating the coronary vessels, whenever there is need for such compensatory adjustments, may be impaired or totally lost.

Fahrenkamp, like so many other observers, rejected the old term of "pseudo-angina" or "angina pectoris vera and falsa" and wrote:

> There are intra-vitam all possible transitional stages from the slightest disturbance of function in coronary circulation to the gravest pathological anatomical changes of the coronary vessels.... next to physical exertion psychic excitement is most likely to precipitate an attack. The greatest sense of annihilation with fear of death occurs chiefly in the second group, because psychic excitement so often produces the first attack, patient and physician may be inclined to consider the condition as "nervous" if the organic findings are slight ....but even the non-neurotic patient may retain such a degree of fear of death from his first attack that he is unable to free himself from a secret anxiety.[3]

Master, Jaffe, and Field[4] stated that hearing of a death, heated argument, shock, hemorrhage, vasovagal syncope, et cetera, may be responsible for an acute attack of coronary insufficiency caused by spasm or inadequate dilatation of the coronary vessels though some degree of coronary sclerosis and/or narrowing of these vessels may be found. Recovery in these cases is usually complete but it depends to a great extent on the inducing factor and on the degree of any underlying coronary pathology. The condition may be acute and very transient or it may last for many days or weeks in more severe cases. Electrocardiograms in mild cases may show no changes at all and no systemic signs such as fever, leukocytosis, or acceleration of the sedimentation rate will occur. Usually however, transitory RS-T depressions and/or T-wave inversion appear and may persist for days and weeks. Fatalities have been reported in less than 5 per cent of cases of coronary insufficiency and death is probably caused by an abrupt onset of ventricular fibrillation. Post-mortem examination usually reveals subendocardial necrosis or multiple small areas of fibrosis with or without some degree of coronary sclerosis.

East and Oram[5] observed a large number of patients who experienced cardiac pain at rest, lasting for more than half an hour, but this was at first and on more than one occasion accompanied by perfectly normal electrocardiograms. Later transient T-wave changes usually developed but without RS-T segment displacements and, of course, without Q waves. They believed that the cardiac pain and transient T-wave changes were due to prolonged coronary spasm, affecting the left coronary artery much more frequently than the right. They further assumed that the reduction in the caliber of the vessel is not sufficient in degree or duration to give rise to significant myocardial changes. Prompted by these findings the same authors also drew attention to the error of assuming that a patient with precordial pain accompanied by a normal electrocardiogram cannot be suffering from true cardiac pain.

In his discussion of angina, Houston[6] also mentioned the importance of neurogenic stimuli, and he believed that if the spasmogenic aptitude is great even a slight stimulus will suffice to evoke the spasm with its characteristic pain.

More recently Evans[7] discussed 10 distinctive tracings which he found among 1,370 patients whose symptoms suggested cardiac pain. These recordings revealed T-wave inversion in leads CR_1 to CR_4 and often also as far as CR_7 and in L_1. In eight of these 10 cases the tracings reverted to normal, while in the remaining two only slight abnormalities persisted. Evans too mentioned the absence of significant Q waves and of RS-T segment deviations and pointed out that when the tracing had recovered, strenuous physical exercise will reintroduce the deformity un-

less a long pain-free interval existed. He suggested that such distinctive tracings of patients subject to cardiac pain should be named "mutable electrocardiograms" and he believed that they indicate coronary arteriospasm with temporary cardiac ischemia rather than lasting coronary occlusion with cardiac infarction. While East and Oram observed that the cardiographic changes always indicated a spasm of the left coronary artery, Evans went a step further and even localized the actual site of the spasm in the left coronary artery, distal to its circumflex branch and proximal to the left marginal branch unless it orginates from the left circumflex artery. He suggested naming this segment of the coronary circulation "which possesses such a special aptitude to spasm 'the coronary floodgate,' for it can suddenly close and interrupt the blood supply to a large area of the heart and as readily it can open and re-establish the circulation." No mention was made of the emotional state of patients who exhibited these "mutable cardiograms."

An interesting observation was made by Berman, Simonson, and Heron[8] who studied the electrocardiographic effects associated with hypnotic suggestions in normal and coronary sclerotic individuals. Hypnotically induced states of anxiety-fear and work pain were studied on 14 normal young people and 11 patients with varying degrees of coronary artery disease. Electrocardiograms were recorded before and during the hypnotic states. In the normal group either type of suggestion produced T-wave changes in 7 cases (50 per cent), elevation in 2 and depression in 5. In the group with coronary artery disease, 4 cases (36 per cent) showed T-wave changes, elevation in 1 and inversion in 3. These results indicate that nervous factors may aggravate an already existing predisposition to ischemic cardiac disease. Observers point out that the emotional state should be considered in interpreting cardiograms in clinically healthy individuals.

Mainzer and Krause[9] reported abnormal electrocardiograms which were taken on people on the operating table immediately before induction of general anesthesia. These "fear recordings" revealed accentuation of already existing changes in patients with cardiac disorders but they also occurred frequently in persons who had had a normal tracing the previous day. The alterations were transient but sometimes they could still be observed 24 hours after operation. The authors classified them in three groups: (1) S-T depression with low inverted or absent T waves; (2) P and T waves high and sharply pointed; and (3) a combination of (1) and (2). These observers believed that the tracings of group 1 indicated a reduced coronary flow due to vagal stimulation, that those of group 2 were due to a sympathetic overactivity, while the appearances of group 3 were thought to be

the result of the interaction of both factors. They suggested that cardiac death on the operating table immediately before the induction of general anesthesia as well as during anesthesia should, at least in some cases, be considered as the extreme outcome of an otherwise "normal" fear reaction.

Werley[10] examined a series of case histories in which the picture presented was that of organic heart disease and where he believed that heart disease was in fact not present. In his opinion it makes no difference if a highly sensitive coronary artery gets into spasm because of emotion or because of an overloaded stomach or physical exertion. The symptoms will always be the same except for the emotional element or its absence. He further believed that even in "true angina" due to coronary sclerosis the attack may be precipitated by emotional excitement and he found that the result is very similar whether coronary arteriospasm is brought about by local organic disease or by overaction of the sympathetic system under the drive of emotion.

Once a person whose emotional reactions are disturbed and who is suffering from precordial pain has convinced himself that his pain is due to serious heart disease, then a new and much more damaging fear is added, namely the fear of imminent sudden death. Various complicated conditioned reflexes, superimposed on this fear and anxiety state, may not develop, and finally it may become extremely difficult to distinguish between these distressing autonomic reactions and underlying heart disease. Tachycardia, transient elevation of blood pressure, dyspnea, perspiration, dilatation of pupils, transient visual disturbances (such as amaurosis fugax), Reynaud's syndrome, et cetera, may all accompany precordial pain and represent vasomotor disturbances associated with an anxiety state.

Scherf and Boyd[11] observed a patient in whom marked pallor of the entire left arm appeared simultaneously with an increase of blood pressure (blood pressure crisis) and angina pectoris. Stimulation of the cervical sympathetic during an attack of precordial pain was observed by Miller and Daley[12] who reported a case which exhibited lacrimation, sweating and vasodilatation of the left side of the face with dilatation of the left pupil and transient ptosis. A voluntary change in the heart rate, particularly acceleration is still a controversial point. It may develop purely voluntarily or be indirectly produced by an emotional state. Voluntary slowing of the heart rate also has been observed in one by merely concentrating on a sad situation.

Stevenson and Duncan[13] described two major patterns of cardiovascular response to stress in individuals with cardiovascular disease. They associated one with mobilization for action, characterized by increased cardiac output as a result of increase in rate and stroke volume, combined with lowering of the pe-

ripheral resistance and slight raising of blood pressure. The second is cardiovascular hypoactivity, diminution in heart rate, stroke volume, and fall of blood pressure. They also observed rapid changes in the electrocardiogram including changes in the amplitude and direction of the T wave occurring in association with changes in the feeling state. Wolf and Wolff[14] were of the opinion that "heart pain in the presence of anatomic narrowing of the coronary arteries may result from increased work of the heart attendant upon prolonged elevation of the blood pressure and cardiac output in association with rage, resentment, anxiety, fear and tension," but they also believed that "heart pain may result from a fall in the cardiac output and coronary blood flow in association with desperation and defeat." They refer to the latter as the "hypodynamic response."

SUMMARY AND CONCLUSIONS

The case of a 41-year-old man, who for almost five years was followed at this hospital for symptoms of progressive psychoneurosis, is reported. Cardiac investigations were persistently negative. The sudden cardiac death of a friend and his subsequent "interest" in heart diseases aggravated his mental condition and unleashed a number of vasomotor symptoms which convinced the patient that death was imminent. He eventually developed true cardiac pain and his electrocardiograms for the first time confirmed myocardial ischemia. The patient died in one of these attacks in bed, but a post-mortem examination failed to reveal coronary occlusion, thrombosis, or myocardial infarction. There was, however, some narrowing at one localized area of the descending branch of the left coronary artery by a single atheromatous plaque. Histologic slides revealed minimal, probably incidental, myocardial fibrosis in the left ventricle.

Some of the literature dealing with the psychosomatic relationship in precordial pain and the changes in the electrocardiograms of emotionally disturbed people, as well as the electrocardiographic findings in coronary spasm and coronary insufficiency, is discussed. If one considers the history of this case and its unusual and bizarre symptoms, there can be little doubt that a severe progressive psychoneurosis existed. For almost five years his mental disturbance was manifested at first by hypochondriacal complaints, but later, after he had heard of the sudden death of a friend and after his subsequent interest in heart diseases, his condition deteriorated into a state of permanent anxiety and fear.

These emotional stimuli affected his autonomic nervous system and resulted in a variety of vasomotor symptoms, such as palpitation, extrasystoles, tachycardia, fluctuation of blood pressure, flushing of his face, dilation of his pupils, sweating, and cyanosis of the hands. While one was perfectly justified in regarding all

these manifestations as "functional," the patient became more and more inclined to regard them as certain proof of an existing and rapidly progressive cardiovascular disease. A vicious circle therefore developed and fear of imminent and sudden death was added to his anxiety state and aggravated his condition considerably.

It cannot be overlooked, however, that a few weeks before his death typical cardiac pain developed and became so severe that a status anginosus existed. This and the eventual fatal outcome will have to be explained by the rather sparse but not altogether negative autopsy findings. The localized small area of narrowing of the lumen of the descending branch of the left coronary artery, caused by an equally small and localized but bulging atheromatous plaque in the absence of any significant sclerosis in the rest of the coronary tree, must have caused a state of chronic coronary insufficiency which eventually led to his death. While under basal conditions the myocardium could apparently still be adequately perfused and oxygenated, the patient's anxiety state with its subsequent imbalance of the vegetative nervous system impaired the normal compensatory adjusting mechanism of the heart and rendered the coronary circulation inadequate for the increased requirements of the myocardium. The coronary reserve seriously diminished and eventually became exhausted.

Whether or not superimposed coronary spasm also played a part during the spasmodic attacks of precordial pain is almost impossible to say. If one postulates inversion of T waves without RS-T segment deviations as the only pathognomonic electrocardiographic findings in coronary spasm, as East and Oram believed and Evans more recently emphasized, then coronary spasm could not be proved in this case. It must, however, be mentioned that all the tracings were taken after and none during an attack of pain. The fatal onset which awoke the patient from his sleep might have been provoked by an anxiety dream and terminated probably with ventricular fibrillation.

ACKNOWLEDGMENT. The author wishes to thank Dr. William Evans for his encouragement to publish this case and for his helpful and constructive criticism.

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IMPLICATED: 22 BILLION PACKS OF CIGARETTES

"Some persons and groups have accepted the information now available on cigarette smoking and cancer of the lung as sufficiently convincing to implicate smoking as the major cause of the cancer and to justify proceeding with active campaigns to inform the public of the dangers of smoking. (Such information as the U. S. Public Health Service has received to date did not prevent the 1957 sales of cigarettes from reaching an all-time high of 22 billion packs.) If smoking does cause cancer it is evident that such campaigns are a necessary responsibility of public health agencies. In view of the numerous unresolved possibilities of bias in the case history population studies, the conflicting epidemiological evidence and the inconclusive animal experimentation, the evidence is still inadequate for moral certainty of the truth of the causal relation. Investigation must continue and definitive interpretation should be postponed."

—EDITORIAL
in *Southern Medical Journal*
p. 537, Apr. 1957

Constitutional Hepatic Dysfunction in a Family

CHARLES M. WEBER, *Captain, MC, USA*

WHILE recognition of constitutional hepatic dysfunction apparently has little significance so far as its effect on the health of the individual is concerned, its recognition is nonetheless important. Frequently confusion arises with hemolytic disease, disease of the liver, and disease of the biliary tract, resulting in unnecessary restrictions on activities of the patient, unnecessary surgical and medical procedures, denial of insurance, and dismissal from the armed services. In brief, recognition of the condition has such a strong negative value to the patient that greater awareness of the entity is highly desirable on the part of the physician.

This report concerns a patient referred to our clinic for evaluation of jaundice. Members of three generations were studied because intermittent jaundice had been observed.

CASE REPORT

A 21-year-old woman, para 0, gravida 1, was referred from the obstetrical clinic to the internal medicine clinic of this hospital for evaluation of jaundice since birth. The patient stated that she had had the usual childhood diseases with no complications or sequelae. She denied any operations or serious injuries.

Both parents were living and well. They both originated in Ireland, and there was no history of jaundice from either. Patient had had three siblings, the first of which was a boy, who had died at age six weeks as a result of pneumonia. He was said to have been jaundiced. The other two siblings were living and well. The brother, age 17, however, also had had jaundice since early childhood.

The patient did not smoke or drink any alcoholic beverages and she took no medication or drugs of any kind.

The review of systems except for the present complaint was completely within normal limits.

From Ireland Army Hospital, Fort Knox, Ky. Dr. Weber is now at Lahey Clinic, Boston, Mass.

The onset of the patient's jaundice condition dated back to several days after birth. She had been thoroughly examined at several institutions and later reassured that her chronic jaundice was of no clinical significance to her. Her only complaints over the years had been moderate fatigue and lassitude. She had never been anemic and there was no history of easy bruising, bleeding, or hemorrhages. There was no history of nausea, vomiting, abdominal pain, diarrhea, or fever. There had been no significant change in the color of the urine or in the stools in the past.

The routine physical examination findings other than scleral icterus and the tumor of pregnancy were perfectly normal. There was no evidence of any liver palms, spider angiomata, or enlarged liver or spleen.

The laboratory data revealed a red blood cell count of 5,000,000 per μl with a hemoglobin of 13 grams per 100 ml and a white blood cell count of 8,800 per μl with a normal differential. The blood morphology and urinalysis were within normal limits. The reticulocyte count was 0.7 per cent. The Coombs test, sickle cell preparation, and serologic test for syphilis were negative. The serum bilirubin was 5.4 mg per 100 ml, with 0.2 mg direct van den Bergh and 5.2 mg per 100 ml indirect van den Bergh. The total cholesterol, alkaline phosphatase, cephalin-cholesterol flocculation, thymol turbidity, and sulfobromophthalein sodium tests were all normal. Urine and fecal urobilinogen studies were perfectly normal. Repeated laboratory studies consistently demonstrated an elevated serum bilirubin, only the indirect component being abnormal. The remainder of the studies remained normal. A liver biopsy demonstrated normal liver architect (figs. 1, 2).

Total evaluation of the data presented led us to the conclusion that this represented a typical case of constitutional hepatic dysfunction.

Physical examination and some laboratory studies were made of the patient's parents, brother, and sister. The mother, who offered no history of intermittent jaundice, revealed a serum bilirubin of 1.4 mg per 100 ml, with 1.2 mg being indirect van den Bergh. The brother was found to be jaundiced but no other physical abnormalities were noted. His laboratory studies revealed a serum bilirubin of 14.1 mg per 100 ml, with a direct bilirubin of only 0.3 mg per 100 ml. Complete liver profile and hematologic work-up were normal. Both the father and sister demonstrated no abnormality whatsoever.

After the termination of the patient's pregnancy, physical and laboratory work-up on her son revealed no abnormality. From the data collected it appeared that there was a familial transmission of icterus on the maternal side of the family.

COMMENT

This condition was first described by Gilbert in 1901.[1] In his investigation into hemolytic jaundice he encountered patients who exhibited a slight degree of bilirubinemia, but lacked all

Figure 1. Photomicrograph of liver with normal lobular pattern and absence of inflammatory or other changes. (× 80)

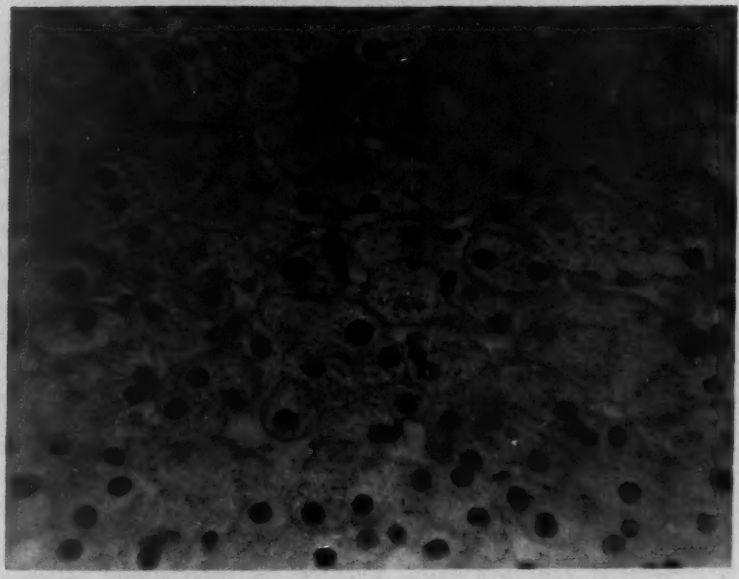

Figure 2. Higher magnification of figure 1. (× 344)

the other symptoms of the disease. He employed the term "cholémie simple familiale." Frequently today, Gilbert's name still remains identified with non-hemolytic, non-obstructive, acholuric jaundice of constitutional type, even though examples of mild hemolytic icterus, hepatic disease and carotinemia were probably included with his original cases.[2] Writers paid little attention to the condition in the next three decades. In 1935 Rozendaal, Comfort, and Snell[3] reviewed cases of jaundice with an "indirect" type of serum bilirubin. Of the cases, 48 showed no evidence of liver disease or hemolytic disease and they considered these patients to fall into the group of constitutional hepatic dysfunction. In 1941 Dameshek and Singer[4] reported two families with this type of jaundice, and coined the term "familial nonhemolytic jaundice." Comfort and Hoyne[5] in 1944 and Meulengracht[6] in 1947 reported series of cases from their own practices.

According to Shay,[7] recent reports "suggest that the defect in bilirubin metabolism in constitutional hepatic dysfunction is caused, in part at least, by impaired activity of glucuronyl transferase, the enzyme responsible for the transfer of glucuronic acid from uridine diphosphoglucuronic acid to the bilirubin."

Constitutional hepatic dysfunction is characterized by chronic or intermittent jaundice which begins during youth or early adult life and lasts for many years. The clinical picture is fairly uniform. The jaundice is seldom intense but it often becomes deeper after intercurrent illness, exertion or fatigue. The patient remains in good health but frequently complains of fatigue, lassitude, dyspepsia, and symptoms of a functional type.[6] Except for icterus, the results of the physical examination are essentially normal. The liver and spleen are not enlarged.

The most important laboratory finding, and of course a prerequisite in the diagnosis, is the bilirubinemia. Serum bilirubin levels are usually under 5 mg per 100 ml although values above 10 to 15 mg per 100 ml have been recorded.[5] The van den Bergh reaction is always of the "indirect variety." Examination of the urine is uniformly negative for bilirubin and increased amounts of urobilinogen. There are no abnormalities of the blood; the sedimentation rate is normal. There is absence of anemia, spherocytosis, reticulocytosis and normal red blood cell fragility, bone marrow, urobilinogen and feces.[4]

Normal values are obtained for liver function studies such as sulfobromophthalein sodium excretion, thymol turbidity and cephalin flocculation. The reaction to injected bilirubin is abnormal. Excretion is greatly delayed and the resultant retention of bilirubin three hours after injection is much greater than the high normal value of 15 mg per 100 ml.

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Gallbladder function is usually normal as judged by cholecystography. Liver biopsy specimens have repeatedly revealed only essentially normal structure.

The etiologic mechanism for this entity appears to be a physiologic defect resulting in the impaired clearance of the serum bilirubin.[2,4] It is now recognized that direct van den Bergh is a glucuronic acid-conjugated bilirubin.[8] Inasmuch as in this condition, there is a failure to form direct reacting bilirubin, the most logical explanation in etiology would be a deficit or disturbance in the enzyme glucuronidase which is necessary for the formation of the conjugated bilirubin and thus producing a high threshold for bilirubin. The defect is said to be a dominant Mendelian trait which may be carried by either sex.[4,9] The fact that several members of the family may be affected is well documented.[2,4,6]

The diagnosis of constitutional hepatic dysfunction is made primarily on the basis of exclusion. The elevations of concentration of bilirubin giving the indirect van den Bergh must be distinguished from those of hemolytic jaundice, chronic hepatic disease, and those persisting after acute hepatic disease. The exclusion of obstructed jaundice and hepatocellular disease by the usual studies and of a subtle hemolytic process by reticulocyte counts and quantitative fecal urobilinogen suggest the diagnosis. A normal liver biopsy gives final histological confirmation.

The variety of this disease as described by Dubin and Sprinz should also be kept in mind. However here, there is an elevation of a direct as well as an indirect type of bilirubin; the presence of dark urine and bilirubinuria; some impairment of liver function studies and nonvisualization of the gallbladder on cholecystography. The liver exhibits normal structure but is grossly and histologically discolored by the presence of a coarse brown pigment in the parenchymal cells.[10]

On the basis of long term follow-up studies, the prognosis is generally said to be excellent. This condition does not predispose to any chronic hepatic disease.[2,6]

Actually, satisfactory treatment has not been devised and it is indeed questionable whether any treatment is indicated if the condition does not affect the health of the individual. Steroid therapy was used in an attempt to reduce the hyperbilirubinemia without satisfactory success.[11]

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USE OF AMPHETAMINE BY ATHLETES

". . . the athlete who will use amphetamine is a problem in mental health as well as a potential problem in physical health. It may be said by some that, since too great a valuation is placed upon the ability to break records of all kinds with a faster mile, broader jump and higher vault, the athlete is under too great popular pressure. Like the paratrooper he may be held to be entitled to reach for a stimulant. However, to equate the long-distance runner in time of peace with the soldier in time of war betrays major loss of perspective. The soldier has his life at stake and the lives of many others. For the disciplined runner the very qualities that make for self-discipline and superior performance are those that should prevent him from taking amphetamine to achieve success. If objective evidence should at any time indicate that stimulant drugs are sometimes being used, the very least of the physician's responsibilities would be to publish the evidence so that watch can be kept for harmful manifestations when they make an appearance in the years to come."

—EDITORIAL
in *New England Journal of Medicine*
p. 1,195, Dec. 12, 1957

Left-Sided Traumatic Diaphragmatic Hernia Involving Solid Viscera

C. WILLIAM ROGERS, *Lieutenant, MC, USN*

THE first account of a diaphragmatic hernia was written by Ambroise Pare[1] in reporting the case of a military officer who died in great pain following a penetrating chest wound. Postmortem examination disclosed the presence of abdominal viscera in the chest. Very little consideration was given to such hernias until World War I, when shrapnel and bayonet wounds of the chest and upper abdomen were common, and many cases of traumatic hernia were seen.

The advent of the roentgen ray was followed by the incidental discovery of diaphragmatic hernias in many patients in whom the condition undoubtedly would have remained undiagnosed unless they came to autopsy. Giffin[2] reviewed the literature in 1912 and found that only about 2 per cent of some 600 reported cases of diaphragmatic hernia were correctly diagnosed before death.

Many classifications have been proposed, but Harrington[3] simply divides all diaphragmatic hernias into traumatic and nontraumatic varieties. Traumatic hernias may result from direct or indirect injury to the diaphragm or from inflammatory necrosis of the diaphragm. The following case is one in which a diaphragmatic hernia, presumably caused by minor trauma, presented a confusing diagnostic problem because of the herniation of solid viscera.

CASE REPORT

The patient was a 49-year-old man who related that he had experienced a tearing sensation, with pain in the lower part of his chest on the left side, after taking a healthy swing with a golf club on 25 February 1957. A roentgenogram of the chest taken on 27 February revealed what appeared to be a mass in the left lower lung field. A thoracentesis, performed at another hospital, yielded 8 ml of bloody fluid.

The patient was transferred to this hospital on 25 March, with a diagnosis of "cystic tumor of left lung," for further diagnostic studies and appropriate treatment. On admission he was entirely asymptomatic.

From U. S. Naval Hospital, Bethesda, Md.

Physical and laboratory examinations were essentially negative and a bronchoscopy with biopsy failed to aid in diagnosis.

Roentgenograms of the chest taken on 10 April (fig. 1) showed soft-tissue density, with reactive changes, in the left lower lung field. A thoracotomy was performed on 11 April with preoperative diagnosis of "undetermined, lung tumor," and the spleen and a large mass of omentum were found in the left thorax, entering through a posterolateral defect in the left diaphragm. The spleen was removed, the omentum was replaced in the peritoneal cavity, and the diaphragmatic defect was repaired.

The patient was discharged from the hospital on 28 April following an uneventful recovery.

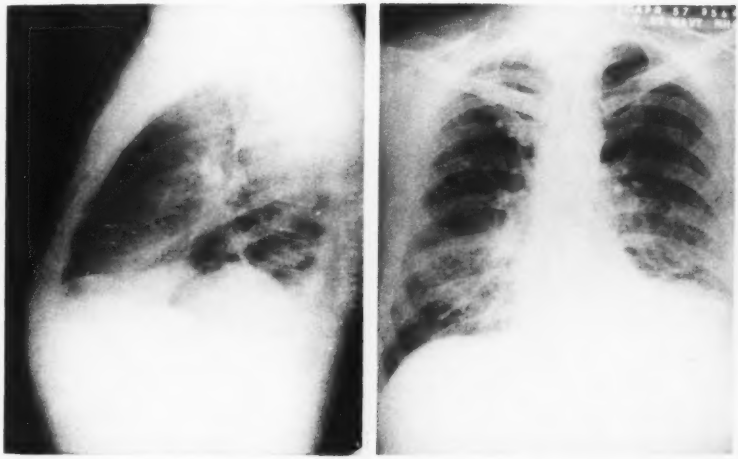

Figure 1. This examination was performed 44 days after the patient experienced pain in the left lower chest while swinging a golf club. The soft-tissue density in the left lower lung field consists of spleen and omentum, and simulates an intrathoracic tumor. Note the reactive changes about the density in the PA projection.

DISCUSSION

When a hollow viscus is herniated through the diaphragm, the diagnosis usually is easy because of the history of trauma and the occasional presence of an air-containing organ above the normal diaphragmatic level. The use of gastrointestinal contrast studies usually will resolve any doubts in this type of hernia. It must be remembered, however, that a diaphragmatic hernia may occur many years after the traumatic incident.[4-6]

The present case is of interest because of the insignificant amount of the trauma that produced the herniation, and also because one expects to find a hollow viscus herniating, when

the diaphragmatic rupture occurs on the left. A number of reports have appeared in recent English literature of cases of liver herniation on the right without accompanying hollow organs,[7] but few cases of solid herniations on the left have been seen. Hughes and associates[8] reported a series of 28 traumatic diaphragmatic hernias, of which 23 were on the left and 3 involved only solid viscera. Incidentally, these three patients remained undiagnosed until thoracotomy.

If the clinician or radiologist suspects a diaphragmatic hernia, diagnostic pneumoperitoneum may be of great value in making a positive diagnosis and in averting a diagnostic thoracotomy. Clay and Hanlon[9] presented four cases of solid visceral herniation through the diaphragm that were diagnosed with pneumoperitoneum without resorting to a diagnostic thoracotomy. They mentioned no history of trauma to any of the four patients, but as illustrated in the case presented here, the traumatic incident may be relatively insignificant and in some cases may only be mentioned on careful questioning.

The roentgenologic appearance of these lesions can be confusing because of the presence of fluid adjacent to the mass in the chest. Thoracentesis may yield bloody, foul-smelling fluid[10] and it is conceivable that puncture of the hernial contents could occur during the procedure, adding further importance to the urgency of establishing a correct diagnosis.

SUMMARY AND CONCLUSIONS

A case of solid visceral herniation through the left diaphragm following minimal trauma was only diagnosed at thoracotomy for suspected lung tumor. Such herniation of solid viscera on the left side is more confusing than that on the right, because one expects to find stomach, colon, or small bowel either accompanying the solid viscus or comprising the hernial contents exclusively. Although not utilized in this case, diagnostic pneumoperitoneum usually will result in a definitive diagnosis. The physician should consider the possibility of a diaphragmatic herniation in any patient whose chest roentgenogram displays a density adjacent to the diaphragm.

ACKNOWLEDGMENT: The author is grateful to Capt. Robert B. Brown, MC, USN, for help in the preparation of this paper and in describing the findings at thoracotomy, which he performed.

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COONSKIN COAT, 1958 MODEL

"A device that will generate variable quantities of heat between the limits of 0 and 1500 B. T. U.'s; provide power for heat distribution; operate at subzero temperatures in moderate winds; permit individuals wearing the outfit to walk about, work with hands, ride or drive unheated vehicles, and be independent of appendages such as air hoses or power connections. The device should operate a minimum of eight hours without refueling; permit rapid removal; be safe to use without creating a fire hazard, uncomfortable hot spots, or toxic fumes; and be, above all, practical for use by individuals. Preliminary studies show that inventive genius is needed to produce a nonelectrical system of this nature or a very light, silent, portable electrical power unit. The fuel must be high energy and, preferably, that normally available to the combat soldier. Heat may be applied to the torso to partially counteract heat losses from the hands and pedal extremities. The critical safe upper limit at skin surface is less than 100°F. While the system described is the ultimate required, solutions to any part of the problem will be welcome.

—INVENTIONS WANTED BY THE ARMED FORCES
in *Missile Design & Development*
p. 22, Dec. 1957

Juvenile Fibromatosis

THEODORE R. WAUGH, *Captain, USAF (MC)*

THE classification of fibrous-tissue tumors has been confused in the past by the large number of specialists who described conditions arising within their own fields.[1] In 1954 a significant contribution was made by Stout[2] toward the organization of this subject, in order to have some framework on which to base a prognosis and evaluate treatment. The juvenile fibromatoses include three more common types; (1) desmoid fibromatoses, the scar-like tissue such as is found in the torticollis of infants; (2) differentiated fibrosarcomas, or those which do not usually metastasize; and (3) fibromatoses, those fibrous growths which cannot be listed elsewhere. In addition, there are five other types, plantar and palmar fibromatoses such as Dupuytren's; juvenile aponeurotic fibroma; keloid fibromatosis; diffuse muscular fibromatosis; and generalized fibromatosis, the multinodular "congenital fibrosarcoma."[3] The separation of these tumors in children, from birth to 15 years, is arbitrary;[2] however, there is evidence that some of the fibromatoses may be congenital,[4] somewhat less than half of reported cases, particularly the desmoid type, manifesting themselves in infancy. Certainly, the fibrous-tissue growth potential is altered in children.[4]

The role of trauma and estrogenic imbalance are considered in certain cases to be of etiologic significance, but a correlation is more frequently absent than present.[5-7]

The following is a case of juvenile fibromatosis with keloid formation in the excision scar.

CASE REPORT

A 16-month-old Caucasian boy had a history of a gradually increasing mass in the lower right lateral thigh since the age of 3 or 4 months. It was the opinion of the mother that in the past 3 months the mass had been increasing more rapidly in size. There was no suggestion of antecedent trauma.

Past History. The patient was born of a normal, full term pregnancy with spontaneous delivery in cephalic presentation. There was one other sibling, a 4-year-old male. In the neonatal period there were no

From 1710th U. S. Air Force Hospital, Donaldson Air Force Base, S. C.

convulsions, hemorrhages, cyanosis, or jaundice noted. The child's development and nutrition were normal.

Physical Examination. The examination was normal in all respects with the exception of a hard, relatively immobile, mass, on the lateral aspect of the lower right thigh which measured approximately 6 cm in its greatest diameter. There was no inguinal adenopathy.

Laboratory Studies. Hemogram prior to admission revealed a leukocyte count of 5,050 per μl with a differential count of 12 per cent neutrophils and 88 per cent lymphocytes. The hemoglobin was 11.8 grams per 100 ml. The bleeding and coagulation times were normal. On hospital admission, the leukocyte count was 6,500 per μl, with a differential count of 8 per cent neutrophils, 88 per cent lymphocytes, 3 per cent monocytes, and 1 per cent eosinophils. Hemoglobin was 10.8 grams per 100 ml. Urinalysis was normal.

Radiologic examination prior to hospitalization revealed the presence of a soft tissue mass in the right lower lateral thigh. This mass was considered to be independent of the periosteum of the femur. There was no calcification.

Course in Hospital. On the day after admission, under general anesthesia, the tumor mass of the right vastus lateralis muscle was exposed. This was estimated to measure 6 by 4 by 2 cm. Because of the absence of encapsulation with infiltration into the surrounding muscle, biopsy alone was performed without any attempt to remove the tumor. The specimen was yellow-white in color and firm in consistency. It was sent for pathologic examination and reported by different examiners as desmoid tumor and reactive fibrosis. Neither pathologist believed there was any evidence of sarcoma (figs. 1 and 2).

Consequently, under tourniquet, the previous biopsy site with the adjacent tumor tissue was excised from the right vastus lateralis muscle, removing a surrounding zone of normal muscle, adjacent tendon, the periosteum of the femur, and the lateral suprapatellar pouch of the knee. Repair was effected by closure of the suprapatellar bursa, reapproximation of the muscle and tendon. The lower extremity was immobilized in a posterior splint with the knee in extension. The patient's postoperative course was uncomplicated and on the 14th hospital day he was discharged in a high leg-cast. In three weeks the cast was removed and the incision was well-healed.

At the five-month follow-up visit the child had regained completely normal knee function, and there was no evidence of recurrence at the site of the operation, or of the development of tumors in other areas. However, the surgical scar showed some definite keloid development.

Pathologic Findings. Grossly, the tumor measured 6 by 4 by 2 cm. It was yellow-white in color and of firm, fibrous-like consistency. There was noted an indefinite line of demarcation from the surrounding muscle tissue. Microscopically, there was a poorly circumscribed mass of

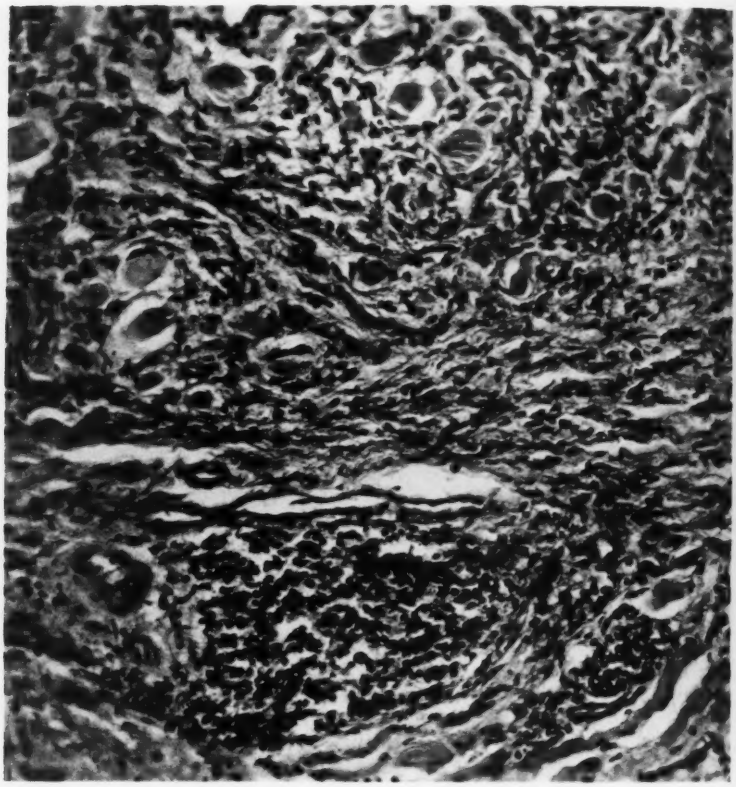

Figure 1. Photomicrograph of section from the biopsy specimen showing cellular exudate. (× 200)

relatively cellular fibrous tissue with slim collagen fibers. There were occasional groups of uniform, slightly hyperchromic fibroblasts, resembling those in the fibrous stroma. Mitotic activity was not noted. Within the tumor tissue were some degenerating muscle fibers with essentially normal muscle adjacent to the tumor tissue. The microscopic picture was one of a juvenile fibromatosis.[6]

DISCUSSION

The juvenile fibromatoses represent an important category of fibrous tissue tumors because of their insidiously progressive growth characteristics.[4] While locally invasive and possibly crippling, they rarely metastasize.[6]

With this rationale, the present treatment is one of radical local excision where this can feasibly be performed.[2,5-6] Stout[2]

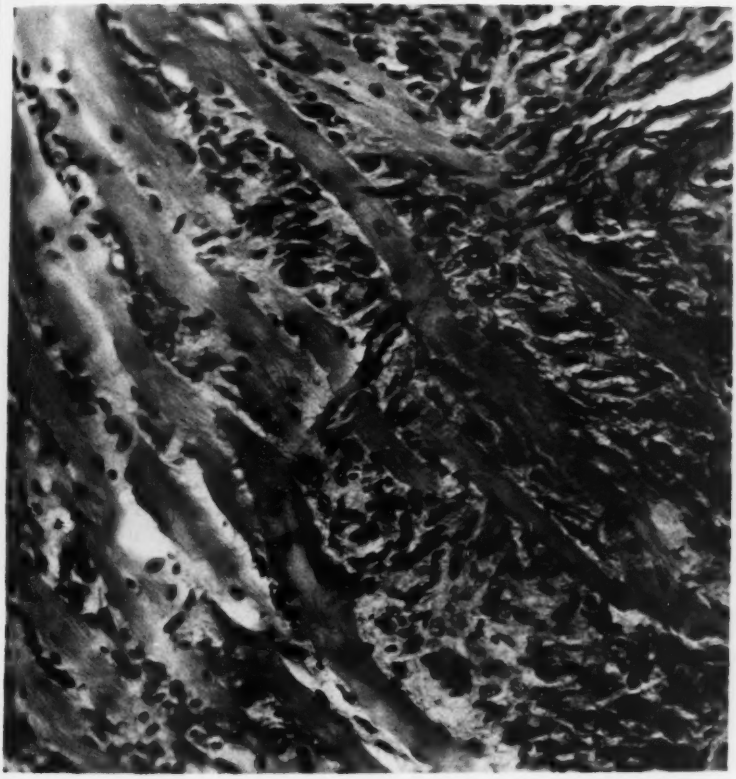

Figure 2. Photomicrograph of section from the biopsy specimen showing infiltration into muscle. (× 250)

noted that some fibromatoses became quiescent after mere biopsy and he suggested that the plantar fibromatoses and the diffuse muscular fibromatoses should be left untreated. However, the tendency to local recurrence is particularly strong in the major groups,[4, 7] and this may necessitate repeated local excisions, which in the extremities is preferable to amputation.

The development of a keloid in the surgical scar represents an interesting association within the fibromatoses and is suggestive of common causative factors.

SUMMARY

A case of juvenile fibromatosis has been presented with the gross and microscopic pathologic findings. This was treated by wide surgical excision of the tumor. The characteristic behavior

of infiltration and tendency to local recurrence, but not metastasis, is the basis for the surgical treatment of these lesions. The development of a keloid, another type of fibrous growth within the juvenile fibromatoses, following excision of the original tumor, is an unusual occurence.

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TREATMENT OF ACNE

"Acne vulgaris, the bogey man of youth, is a benign disease in itself, commonly encountered, refractory to treatment, aesthetically unpleasing, demoralizing, with a complex etiology and pathogenesis. No specific treatment is known. Good health habits and local antisepsis are the basis of treatment. The efficiency of radiotherapy is undeniable. In a series of 107 patients, the association of multi-vitamin preparations in high dosage with thyroid extract and antihistaminics both in small doses has given . . . excellent results in 58 per cent of cases and satisfactory results in 90 per cent. This therapy on account of its very simplicity, harmlessness and efficacy constitutes a treatment of choice in acne vulgaris."

—PAUL POIRIER, M. D.
in *Canadian Medical Association Journal*
pp. 869-870, Nov. 1, 1957

Ureteral Ectopia

ANTHONY A. BORSKI, *Major, MC, USA*
ROBERT W. PARSONS, *Captain, MC, USA*

WE believe that there is a widespread lack of awareness of ureteral ectopia. This is borne out by the fact that 26 different physicians treated a young girl during a seven-year-period without suspecting this congenital condition as a cause of her repeated illnesses.

CASE REPORT

An 11-year-old girl was admitted to this hospital on 16 October 1957 for urologic work-up for urinary incontinence. The history, as obtained from the parents, dated the child's difficulties for approximately 8 years. At age 3, the child had intermittent low-grade fevers associated with dysuria, and her underclothing and bed were almost always wet. In addition, there was a yellowish vaginal discharge. She was treated with various medications with some improvement. However, she continued wetting her underclothing almost continuously. After several years of treatment with medications and after having been treated by a dozen different physicians for the urinary incontinence, it was suggested that psychiatric treatment should be used in order to help control the symptoms. After a course of psychiatric treatment, there was no improvement and the mother continued to seek medical aid for this distressing problem in this young child. Different physicians were consulted again and vaginal douches and suppositories were used along with antibiotic therapy. After seven years of frustrating efforts the 27th physician suggested a urologic investigation. This child had never previously had a urologic work-up. One physician even had suggested psychiatric treatment for the mother because of her insistence that this was not an emotional problem as she believed that she knew her daughter better than the physicians.

Physical examination was essentially normal except for inflammation and lichenification over the genitalia and inner thighs from urinary leakage.

Urinalysis, complete blood cell count, and blood urea nitrogen were all normal. A tuberculin skin test using PPD was negative in 48 hours. Roentgenographic findings of the chest were normal. Intravenous urograms showed a reduplication of the right kidney and a normal left kidney (fig. 1).

From Fitzsimons Army Hospital, Denver, Colo.

On 22 October, under Pentothal (brand of thiopental sodium) anesthesia, a cystoscopy was performed, and this showed a normal bladder with a single orifice on the right and a normal left orifice. During this examination a catheter was passed up the lower right kidney which

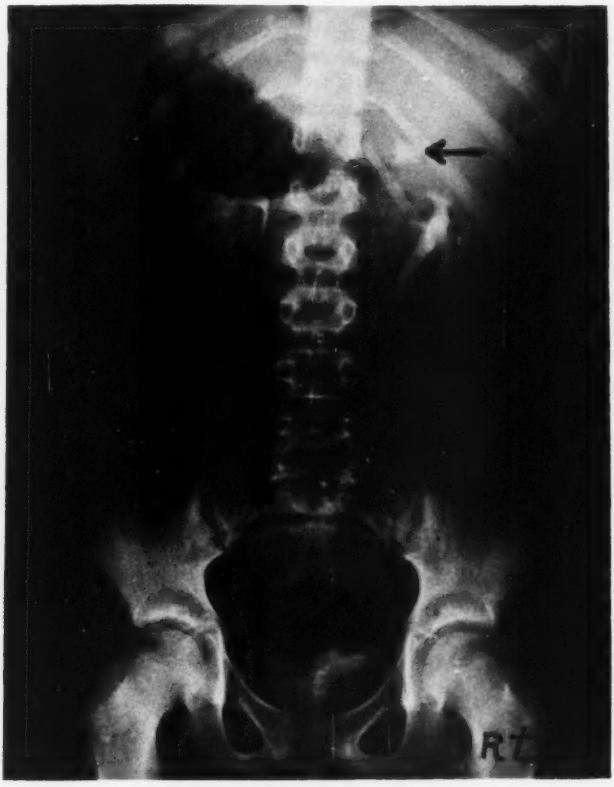

Figure 1. Intravenous urogram. The arrow points to the superior segment of the double kidney on the right. The lower kidney is normal.

was normal. A vaginal examination revealed an ectopic ureteral orifice in the anterior vagina. A catheter was introduced through the vagina into this ectopic orifice and it became coiled in a dilated tortuous ureter (fig. 2). On 25 October a small upper kidney and markedly dilated, tortuous ureter were removed. The lower kidney was normal. Postoperatively there was no further urinary dribbling or discharge from the vagina. A 6-month follow-up revealed a normal intravenous urogram and a happy, asymptomatic child and mother.

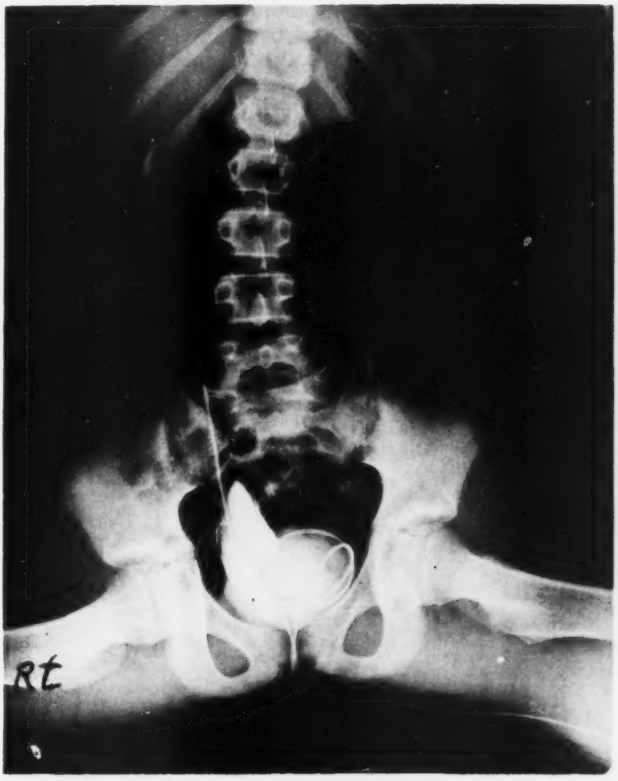

Figure 2. Retrograde urogram. The catheter through the bladder shows a normal ureter. A catheter through the vaginal ectopic orifice is coiled in the tortuous dilated aberrant ureter.

DISCUSSION

Ureteral ectopia is no longer considered a rare entity. It is being recognized clinically with increasing frequency. A review of the literature by Burford, Glenn, and Burford[1] in 1949 showed that 425 cases had been reported since the review of Giviale in 1843; and each of the past 9 years has seen a number of new cases reported. In autopsies in children, the incidence of ureteral ectopia is reported to be 1 in 1,600; and is found from three to four times as frequently in females as in males.[2] However, as illustrated by our case, there is still a widespread lack of awareness of this diagnostic possibility.

The embryologic background was well described by Hepler[3] and by Wesson.[4] The ectopic orifice most commonly drains a reduplicated ureter, and in 75 per cent of the cases it is the

ureter from the superior pelvis that is ectopic. The renal tissue drained by the involved ureter is usually hypoplastic and shows destructive changes as a result of obstruction and infection.

The ectopic orifice may open into the posterior urethra, vas deferens, epididymis, or seminal vesicle in the male, giving rise to symptoms of recurrent pyuria. In the female, the opening is usually in the urethra or introitus adjacent to the external urethral meatus; it also may be found in the vagina, cervix, or uterus. The typical history in the female patient is one of incontinence of urine in the presence of normal bladder function, frequently accompanied by recurrent urinary infection.

The diagnosis is frequently difficult because the renal tissue drained by the ectopic orifice may be nonfunctioning. In the female, a careful examination of the usual sites of the openings is necessary. Intravenous urography, retrograde pyelography, intravenous injection of indigo carmine, and careful cystoscopic examination are necessary to establish the diagnosis. Katzen and Trachtman[5] described the use of the vaginogram as a diagnostic aid.

Treatment consists in following sound urologic principles. In some cases ureterovesical transplantation may permit salvage of remaining functional renal tissue. In the majority of cases, however, heminephrectomy with ureterectomy is indicated. Ligation of the aberrant ureter is inadvisable because of the frequency of infection.

SUMMARY

Ectopic ureteral orifice is not a rare condition. Many more cases will be clinically recognized if all cases of recurrent pyuria in males and of urinary incontinence in females are carefully examined with this condition in mind. The case presented illustrates the common delay in diagnosis resulting from a lack of awareness of this entity.

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NEW MARTIN ARMY HOSPITAL DEDICATED IN CEREMONY AT FORT BENNING, GEORGIA

Built at a cost of $8,000,000, the Martin Army Hospital at Fort Benning, Georgia, was formally dedicated on 1 July 1958. The building has nine stories, contains 500 beds and is completely air conditioned. Constructed along highly functional lines, the new hospital features a centralized oxygen supply, a pneumatic tube message carrier system, the latest audio-visual nurses' call system, and the newest equipment for reception of television for patient use through a master antenna system.

Mrs. Martin, left, Colonel Skinner, General Freeman, and General Cooney.

During the dedication ceremony, Major General Paul L. Freeman, Jr., USA, Commanding General of the U. S. Army Infantry Center, presented the key to the hospital to Colonel Robert B. Skinner, MC, USA, Hospital Commander. Mrs. Joseph I. Martin unveiled a large bronze dedicatory plaque, which is located in the lobby of the main entrance to the hospital, inscribed in honor of her husband, the late Major General Joseph I. Martin, MC, USA. The dedication address of Major General James P. Cooney, MC, USA, Deputy Surgeon General of the Army, is published in this issue of the *Journal.*

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ADMIRAL HOGAN GIVEN PRIVATE AUDIENCE WITH POPE PIUS XII ON EUROPEAN TOUR

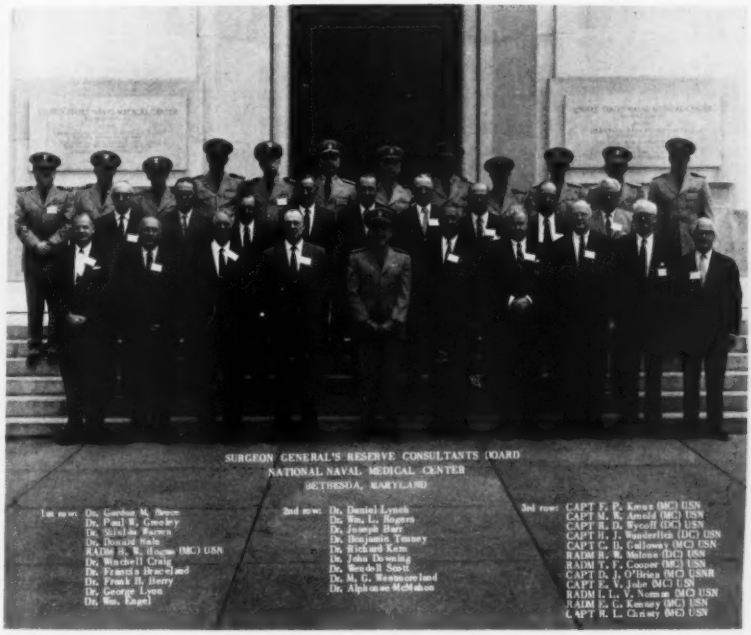
On a recent tour of naval facilities in Europe, Rear Admiral B. W. Hogan, Surgeon General of the Navy, had a private audience with His Holiness, Pope Pius XII. Now 83, the Pope appeared in excellent health according to Admiral Hogan.

Admiral Hogan and Pope Pius XII

"He spoke to me in English," he said, "not only of the importance of the work that doctors of medicine are doing in the military services but emphasized several times the great importance of medicine in promoting international good will, tolerance, and understanding among all nations of the world."

NAVY MEDICAL RESERVE CONSULTANTS BOARD DISCUSSES GRADUATE TRAINING PROGRAM

The Reserve Consultants Board of the Bureau of Medicine and Surgery recently met at the National Naval Medical Center, Bethesda, Maryland, to discuss the Navy's Graduate Medical Training Program with the Surgeon General, Rear Admiral Bartholomew W. Hogan, MC, USN, and his staff. Each member of the Board represents one of the medical specialties, and the majority hold teaching positions in medical schools throughout the country. The Honorable Frank B. Berry, M. D., Assistant Secretary of Defense (Health and Medical) was a guest at the meeting.


SURGEON GENERAL'S RESERVE CONSULTANTS BOARD
NATIONAL NAVAL MEDICAL CENTER
BETHESDA, MARYLAND
1st row: Dr. Gordon M. Brown
Dr. Paul R. Greeley
Dr. Shields Warren
Dr. Donald Wale
RADM B. W. Hogan (MC) USN
Dr. Winchell Craig
Dr. Francis Braceland
Dr. Frank B. Berry
Dr. George Lyon
Dr. Wm. Engel
2nd row: Dr. Daniel Lynch
Dr. Wm. L. Rogers
Dr. Joseph Barr
Dr. Benjamin Tenney
Dr. Richard Kern
Dr. John Downing
Dr. Sendel Sorff
Dr. M. G. Westmoreland
Dr. Alphonse McMahon
3rd row: CAPT F. P. Keuer (MC) USN
CAPT M. K. Arnold (MC) USN
CAPT R. D. Tyroff (DC) USN
CAPT H. J. Wunderlich (DC) USN
CAPT C. B. Gallower (MC) USN
RADM B. W. Mehrtens (DC) USN
RADM T. F. Cooper (MC) USN
CAPT D. J. O'Brien (MC) USNR
CAPT E. V. John (MC) USN
RADM L. L. V. Norman (MC) USN
RADM E. C. Kenney (MC) USN
CAPT R. L. Christy (MC) USN

The Navy's Graduate Medical Training Program was planned and instituted at the close of World War II by several of the members of the Board who were senior officers in the U. S. Naval Reserve, on active duty at that time. Since their release from active duty they have continued their interest in the Navy and have given freely of their time and advice, visiting the Navy's medical facilities at the request of the Surgeon General, to review the training programs. The high standards of the training have produced well-qualified naval medical officers, who in turn provide better patient care. The program has been a very important feature of the professional career pattern of the Navy.

A MESSAGE FROM THE A. M. A.

The American Medical Association has always been keenly interested in, and since World War II particularly active concerning, the medical care furnished our military personnel. Opportunities for study, research, actual treatment of patients, rotation of medical assignments, and assignment of medical personnel to nonmedical duties also are matters of concern to the Association. The development of a strong and attractive medical service staffed for the most part by qualified career officers in the Armed Forces, Veterans Administration, and Public Health Service is, in the Association's view, in the best interest of the public welfare. The development of such services depends on the confidence and intimate collaboration of the medical profession of the nation.

Since the creation of its Council on National Defense in 1946, the Association has been in particularly close and constant contact with the complex problems of military medicine. The best medical care for our servicemen must be provided in a manner that is economical and yet consistent with the health needs of our civilian population. To accomplish this, the Association has consistently advocated that the military medical services be strengthened by legislation and administrative action which would attract and retain well-qualified physicians in numbers sufficient to meet the needs of the military services.

One effort toward this accomplishment is to offer to such qualified physicians a pay scale at least as attractive as that offered by other Federal medical services. In other words, the Association believes that the Armed Forces must be in a position at least equal to that of other Federal agencies if it is to attract and retain medical personnel who will render the highest medical service.

The Medical and Dental Officer Career Incentive Act of 1956 had the unreserved support of the American Medical Association. Witnesses for the Association appeared before the committees of Congress which considered this legislation. This Act is a valuable method of preserving and improving the quality of military medical manpower. This legislation provides special pay of $100 per month which increases in three increments of $50 each at 2, 6, and 10 years of active service as a medical officer for a total special pay of $250. Thus, the career medical officer is recompensed for his personal expenses, tuition, et cetera, in securing his premedical and medical education to a degree commensurate with line officers who attended the military or naval academies. Such step-up salary increases appeal to

From the Council on National Defense of the American Medical Association. The views and opinions expressed are not necessarily those of the Department of Defense. —*Editor*

experienced officers, many of whom had intended to abandon military careers for the private practice of medicine. The result of this legislation has been salutary. The increase in special pay has had the effect of slowing down the resignation rate of regular military medical officers, of increasing the procurement of new officers, and of providing stability in the medical corps.

The benefits achieved by military medicine under the Medical and Dental Officer Career Incentive Act of 1956 were threatened during the legislative process which culminated in the enactment of Public Law 85-422, approved May 20, 1958, which adjusts the method of computing basic pay for officers and enlisted members of the uniformed services. It was proposed that the special pay received by medical officers of the Armed Forces be reduced by as much as $100 to $150 monthly in the groups of the most experienced physicians. True, the proposed reduction would not have resulted in decreased compensation for any medical officer but, nevertheless, the proposal undoubtedly would have had an adverse effect on the attraction and retention of qualified physicians for the military services. For this reason, the Association in vigorous opposition to the proposed reduction for medical officers made its views known to the Congress.

It was, and is, the Association's view that the special pay provisions of the Medical and Dental Officer Career Incentive Act of 1956 is largely a method of equalizing earnings to compensate for the long and expensive medical education and the resultant delay in earnings of the medical doctor.

It is gratifying that the Association's position was accepted by Congress and thus prevailed in this matter. The recently enacted Public Law 85-422 did not alter the incentive pay provisions of the Medical and Dental Officer Career Incentive Act of 1956.

The military medical officer may be certain that the American Medical Association, through its Council on National Defense, will continue to support legislative proposals that contribute to the desired goal of a stable career medical corps, which is essential if the members of the U. S. Military Forces are to receive medical care that is second to none.

World Congress of Aviation Medicine

Brigadier General M. S. White, USAF (MC), president of the Aero Medical Association, will officially represent the society and the Surgeon General of the Air Force at the World Congress of Aviation Medicine in Louvain, Belgium, 23-27 September.

SECOND ARMY CONSULTANTS AND HOSPITAL COMMANDERS MEET AT FORT GEORGE MEADE

A conference of civilian medical consultants and hospital commanders of the Second United States Army was held at Fort George G. Meade, Maryland, on 28-29 May 1958, under the direction of Colonel Francis P. Kintz, MC, USA, Second Army Surgeon. The U. S. Army Hospital, Fort Meade, under the command of Colonel Charles S. Mudgett, MC, USA, was the host facility.

Drs. Ravdin and Guttmacher, and Colonel Kintz.

The purpose of the conference was to further the professional relationships between civilian medical consultants and Second Army hospital commanders, to evaluate and discuss recent advances in medical procedures, technics, and drugs, and to appraise the professional standards of medical care within the Army's treatment facilities. In addition to the hospital commanders who attended, distinguished guests included Major General I. S. Ravdin, MC, USAR (Ret)., Chairman, Second Army Civilian Consultants Advisory Committee; Colonel Douglas B. Kendrick, MC, USA, Chief Surgical Consultant, Department of the Army; Dr. Manford S. Guttmacher, Chief Medical Officer of the Supreme Bench of Baltimore, Maryland; and Dr. M. R. Hilleman, Director of Virus Research, Merck Sharpe & Dohme Research Laboratories.

WEST POINT HOSPITAL GIVEN ACCREDITATION

The West Point Army Hospital recently was granted accreditation by the Joint Commission of Accreditation of Hospitals. Lieutenant General Garrison H. Davidson, USA (right), Superintendent of the United States Military Academy is shown presenting the announcement of the accreditation to Colonel James B. Stapleton, MC, USA, Hospital Commander.

Official Decorations

The following awards were recently announced by the Department of the Army.

Legion of Merit

Robert T. Gants, Col., MC, USA*

Commendation Ribbon

Esther Clausen, Lt. Col., ANC, USA

Thomas R. Ostrom, Capt., MSC, USA

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*First Oak Leaf Cluster (awarded posthumously)

Army Dedicates New Hospital on Okinawa

Lieutenant General James E. Moore, USA, High Commissioner of the Ryukyu Islands, was the principal speaker at the dedication recently of a new 250-bed Army hospital on Okinawa. Many distinguished visitors stationed in the Far East attended the ceremonies for the only military hospital in the Islands when host for the occasion was Colonel James T. McGibony, MC, USA, Commander of the new hospital and Surgeon, U. S. Army Ryukyu Islands.

New U. S. Army Hospital, Ryukyu Islands

The construction of the hospital began in December 1954, and many obstacles were encountered by the engineers before its completion. The building is partly air conditioned, and contains oxygen and master clock systems, vacuum installations for the operating and delivery rooms, and an elapsed timer system in the surgery. Staffed by Army medical personnel, the new hospital offers its services to all U. S. military units in the Islands.

Captain Gorby Receives Hoff Medal

Captain Earl W. Gorby, MC, USA, son of Major General Alvin L. Gorby, MC, USA, Chief Surgeon, U. S. Army Europe, was the 1958 recipient of the Hoff Medal, awarded annually to the student with the highest proficiency in the Military Medicine and Allied Sciences Course of the Walter Reed Army Institute of Research. Established in 1897 in memory of Colonel Alexander H. Hoff, the medal formerly was given each year to the outstanding graduate of the Army Medical School. Graduation ceremonies of the nine-month course were held on 20 June.

OFFICERS CERTIFIED BY SPECIALTY BOARDS

Supplementary Listing

The Surgeons General of the military departments have announced certification by specialty boards of the following officers of the regular establishment since publication of listings in previous issues of the *Journal*.

American Board of Radiology

Edward A. Sheldon, Capt., USAF
Matthew F. J. Yenney, Jr., Lt., USN

American Board of Obstetrics and Gynecology

Robert C. Drips, Lt. Comdr., USN
Halvdan G. K. Faaland, Comdr., USN
Stratton R. Easter, Maj., USAF
Mable A. Frew, Lt., USN

American Board of Internal Medicine

Robert E. De Forest, Lt., USN
Thomas W. Sheehy, Capt., USA
Max E. Musgrave, Lt. Comdr., USN
James L. Spencer, Capt., USN

American Board of Surgery

Harry C. Green, Jr., Lt. Col., USAF

Board of Thoracic Surgery

Billie G. Streete, Lt. Col., USAF

American Board of Anesthesiology

Robert J. McCann, Maj., USAF

American Board of Preventive Medicine

Public Health

Henry J. Donnelly, Maj., USA
Alois Peczenik, Maj., USA

Aviation Medicine

Charles N. Moss, Lt. Col., USAF
Lawrence T. Odland, Maj., USAF
Henry C. Moritz, Jr., Maj., USAF

Occupational Medicine

John A. Peterson, Maj., USAF

American Board of Oral Surgery

John P. Jarabak, Capt., USN
Jerome C. Stoopack, Capt., USN

American Board of Periodontology

Peter C. Fedi, Lt. Comdr., USN

American Board of Veterinary Public Health

Joseph D. Manges, Col., USA

Dr. Youmans Named Medical Research Advisor

Dr. John B. Youmans, dean of Vanderbilt University Medical School, has been appointed Technical Director of Research for the Surgeon General of the Army, effective 1 September 1958. He is resigning his position at Vanderbilt, where he has served since 1950 to move to Washington. A retired colonel of the Army Medical Corps, Dr. Youmans served during World War II in the CBI, Pacific and European Theaters, and was awarded the Legion of Merit and the French Legion of Honor. He will serve as principal advisor to the Surgeon General and his staff in all scientific and technical matters related to medical research and development.

Dr. Youmans

Dr. Youmans is now serving as consultant to the Army Surgeon General's Preventive Medicine Division and to the Interdepartmental Committee on Nutrition for National Defense. He is a member of the Assistant Secretary of Defense's Panel on Military Medicine and the National Research Council Committee for the Quartermaster Subcommittee on Nutrition.

Col. Snyder Heads Physical Therapists

Lt. Col. Agnes P. Snyder, Assistant Chief of the Army Medical Specialist Corps and Chief, of its Physical Therapist Section, recently was elected president of the American Physical Therapy Association for a three-year term at the 1958 convention of the organization at Seattle, Washington. She is the retiring Speaker of the House of Delegates of the association. Colonel Snyder who is a native of St. Mary's, West Virginia, is a graduate of Marshall College, Huntington, West Virginia, the physical therapy course conducted by the Army Medical Service at Walter Reed Army Hospital, Washington, D. C., and holds a master's degree in physical therapy from the Medical College of Virginia. She entered the Army service in 1933.

Colonel Snyder

DEATHS

CANADA, Joseph Clayton, Lieutenant Colonel, MC, USAR, of Tahlequah, Okla.; stationed at Ft. Bragg, N. C.; graduated in 1929 from the University of Oklahoma School of Medicine, Oklahoma City, Okla.; commissioned a lieutenant colonel in the United States Army Reserve 28 May 1953; ordered to active duty 5 July 1953; died 14 June 1958, age 54, at Walter Reed Army Hospital, Washington, D. C., of a heart attack.

COX, Ardis Tillman, Lieutenant Colonel, MSC, USA, of Long Beach, Calif.; stationed at William Beaumont Army Hospital, El Paso, Tex.; graduated in 1932 from the University of New Mexico, Albuquerque, N. M.; commissioned a first lieutenant in the Army of the United States and ordered to active duty 12 August 1943; died 28 May 1958, age 48, at El Paso, of myocardial infarction.

MANN, Joseph Lee, Lieutenant Colonel, USAF (MC), of Hampton, Va.; commander of the U. S. Air Force Hospital, Griffiss Air Force Base, New York; graduated in 1938 from the University of Virginia Department of Medicine, Charlottesville, Va.; commissioned a first lieutenant in the Army of the United States 26 April 1942; ordered to active duty 11 May 1942; ordered to inactive duty 2 January 1946; transferred to the United States Air Force Reserve and ordered to active duty 10 January 1949; commissioned a captain in the United States Air Force 10 August 1950; died 19 March 1958, age 43, at the U. S. Air Force Hospital, Langley Air Force Base, Virginia, of myocardial infarction.

MARCANTONI, Charles Louis, Lieutenant, DC, USN, of Jackson Heights, N. Y.; stationed at the Naval Communication Facility, Port Lyautey, Morocco; graduated in 1957 from the Georgetown University School of Dentistry, Washington, D. C.; appointed a lieutenant in the United States Naval Reserve 1 July 1957; ordered to active duty 15 July 1957; appointed a lieutenant in the United States Navy 27 May 1958; died 15 June 1958, age 28, at Mehdia Beach, near Port Lyautey, of drowning.

THAYER, James Manning, Jr., Captain, USAF (MC), of Memphis, Tenn.; stationed at the U. S. Air Force Hospital, Landstuhl, Germany; graduated in 1954 from the University of Tennessee College of Medicine, Memphis, Tenn.; commissioned a first lieutenant in the United States Air Force Reserve in 1955, ordered to active duty 24 February 1955; died 15 February 1958, age 27, at Mount Vesuvius, near Naples, Italy, in a military aircraft accident.

THOMPSON, Henry Autry, Lieutenant Commander, MSC, USN, of Willisville, Ark.; stationed at the U. S. Naval Hospital, Guantanamo Bay, Cuba; enlisted in the United States Navy 8 October 1931, and served in an enlisted status until 15 May 1943; appointed a warrant officer in the United States Navy 15 May 1943; appointed ensign (HC) in the United States Navy 15 July 1944; died 28 May 1958, age 45, at the U. S. Naval Hospital, Guantanamo Bay, Cuba, of a heart attack.

Army Chemical Corps Develops New Mask

The Army Chemical Corps has announced the development of a new type of oronasal air-filtering mask that is said to be 10 to 15 times more effective in filtering out airborne bacteria than the conventional surgical mask. It will be used in testing the efficiency of experimental warfare protective masks.

Reviews of Recent Books

THE CHEMISTRY AND CHEMOTHERAPY OF TUBERCULOSIS, A compilation and critical review of existing knowledge on the chemistry of tubercle bacilli and their products, chemical changes and processes in the host, and chemical aspects of the treatment of tuberculosis, by *Esmond R. Long*, M. D., Ph. D., Sc. D. 3d edition. 450 pages; illustrated. The Williams & Wilkins Company, Baltimore, Md., 1958. Price $12.

This book by an internationally known and leading authority on tuberculosis is a compilation and critical review of existing knowledge on the chemistry of tubercle bacilli and their products, chemical changes and processes in the host, and chemical aspects of the treatment of tuberculosis. The author attempted to correlate the subject matter in such a way as to make the mechanism of action of antituberculosis drugs more clearly understood. The chemical aspects of tuberculosis are so varied and complex that a summation of the known facts concerning tuberculous persons and the chemistry of tubercle bacilli should be helpful to research workers and specialists seeking to find improved methods of treatment of this serious disease. The practical information on chemotherapy of tuberculosis makes this volume a very desirable reference for our medical libraries.

—*CARL W. TEMPEL, Brig. Gen., MC, USA*

PHYSICAL METHODS IN PHYSIOLOGY, by *W. T. Catton*, M. Sc. 375 pages; illustrated. Philosophical Library, Inc., New York, N. Y., 1957. Price $10.

In no sense is this a textbook of physiology; it is instead a compact and well-written volume on physical methodology, discussing for the most part technics unavailable to the medical student during his preclinical training. Mathematical formulation is extensive, and a theoretical basis for described measurements is usually presented. With this emphasis, there is minimal discussion of data and even less on correlation.

As might be expected, best coverage is given to muscle, nerve, and the circulatory system; there is no direct approach to endocrines or digestion. A concise but helpful section on relevant electronics is included.

Though conspicuously shorter than *Glasser's Medical Physics*, this text should be of value to the physiologist or to the research worker concerned with physical methodology. While medicine moves ever closer to a physicochemical basis, we have not yet reached the era whe e this volume will be of interest to the clinician.

—*ROBERT D. DRUYAN, Lt., MC, USNR*

THE STORY BEHIND THE WORD, Some Interesting Origins of Medical Terms, by *Harry Wain*, M. D. 342 pages. Charles C Thomas, Springfield, Ill., 1958. Price $8.50.

This is an alphabetical compilation of more than 5,700 words with a brief statement of the origin and history of each term. Also included are brief biographies of great personalities of medicine. While the

author, a physician, states in his introduction that these are medical terms and that his concern is with medical etymology, there are a great number that appear to have little meaning within the medical frame of reference. For example, one wonders about the inclusion of such words as climate, geography, blockhead, idea, cynic, oyster, watershed, pen, jerry-built, and biscuit.

The Story Behind the Word is interesting, but it falls somewhat short of the claims of the author who calls it a "veritable treasure house of linguistic adventure" which gives medical terms "a newer and fuller meaning." Perhaps the answer lies in his further statement that the project was undertaken as a hobby and involved "painstaking research" over a 10-year period—which obviously culminated in some 5,700 terms now published in a volume. One believes that either the volume should have been edited more systematically to include only medical terms, or else not described as a reference work on medical etymology. The casual reader will find the style witty and the reading interesting, but the volume is a luxury item for the library; it cannot be considered a basic reference. —*MAE M. LINK, Ph. D.*

THE ESSENCE OF SURGERY, by *C. Stuart Welch*, M. S., M. D., Ph. D. and *Samuel R. Powers, Jr.*, A. B., M. D., M. Sc. D. 320 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $7.

This is a concise and engaging treatise on the basic principles and foundations of present surgical practice. After a short historical review which sets the stage for the basic concepts which follow, the authors have proposed that the surgeon's entire area of practice can be categorized into three types of injuries. They are loss of body tissue (wounds), loss of body fluids, and infection. The development of their thesis on these broad concepts is logical, lucid, and comprehensive, despite the small size of this volume. The philosophic approach of the authors truly expresses the essence of surgery. The broadening experience resulting from perusal of this volume is extremely gratifying, and is recommended particularly to the younger man beginning his surgical training. Here, he may consciously view early in his career the foundations of a discipline which will engage his entire lifework. For the trained surgeon, this volume will integrate many isolated surgical experiences into a composite, orderly structure worthy of much thoughtful contemplation. I believe the authors have been unusually successful in presenting their thesis. This volume is highly recommended for the surgeon's bookshelf.

—*DAVID GOLD, Col., USAF (MC)*

HANDBOOK OF TREATMENT OF ACUTE POISONING, by *E. H. Bensley*, M. B. E., B. A., M. D., F. A. C. P., and *G. E. Joron*, B. A., M. D., C. M., F. A. C. P. 2d edition. 212 pages. Published by E. & S. Livingstone Ltd., Edinburgh and London, 1958. The Williams and Wilkins Company, Baltimore, Md., exclusive U. S. agents. Price $4.

This small, concise book is presented as a quickly available, specific guide to the immediate treatment of acute poisonings. It is not intended to be a textbook of toxicology. The book is small enough

to be carried easily in a physician's bag or even a coat pocket, and probably would best serve its purpose by being accessible for quick perusal when needed.

The book is divided into two sections, the first devoted to principles of treatment; this is a good review of the subject. The second section deals more specifically with various types of poisons. Recommended treatment in each instance includes measures to be used both before and after the arrival of the physician.

The book fulfills its stated purpose very well, and should be of special value to house officers and physicians engaged in general practice.—*HARVEY E. REITZ, Capt., (MC), USN*

OBJECTIVE APPROACHES TO TREATMENT IN PSYCHIATRY, by *Leo Alexander,* M. D. American Lecture Series, Publication No. 327, Monograph in American Lectures in Objective Psychiatry, edited by *W. Horsley Gantt,* M. D. 139 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $4.50.

This is an erudite, stimulating approach to psychiatric treatment. The book is thought-provoking, and demonstrates considerable research and thinking, much of it original. It attempts to explain empirical treatment methods by carefully worked out scientific evidence, some of which has admittedly not been subjected to experimental or clinical validation. The author points out that the history and clinical examination should be augmented by things that can be tested objectively. In this connection, he gives an explanation of the adrenalin-mecholyl test, and describes his use of the test as an indicator of prognosis and potential responsiveness to treatment. An excellent summary of the various types of electrotherapy and of the various newer types of chemotherapy is included. In a chapter on frontal lobotomy the author advocates this form of treatment in some cases. The chapter on psychotherapy involves primarily a discussion of how "working through" compares to Pavlov's laws of the higher nervous activity of the cerebral cortex. This book has merit because, rather than relying on empiricism, it attempts to approach the various psychiatric treatments objectively and give to them a scientific explanation. It is of interest primarily to psychiatrists, and would appeal more to the "organic" or "eclectic" men in the field and probably less to the strictly "dynamically" oriented; yet it is well worth reading as an excellent summary (with an extensive bibliography) of one of the attempts to correlate the observed somatophysiologic factors in psychiatric treatment with the psychic factors.

—*DASIL C. SMITH, Lt. Col., USAF (MC)*

BIOLOGY OF NEUROGLIA, Compiled and edited by *William F. Windle,* Ph. D., Sc. D. 340 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $8.50.

The term neuroglia refers to several highly specialized but prominent cell types found in the nervous system. The first function assigned these cells and indeed the one still described in neuroanatomical

texts is that of serving as the connective tissue of the nervous system —the nerve "glue," in translation. As time has passed it has become clear that the microglia, one of the neuroglial types, acts as the scavenger of the nervous system and is, in fact, derived from mesodermal wandering cells originating from vascular elements. The function of the remaining cell types, the astrocyte and the oligodentrocyte, however, has remained unknown. The conception that these latter function as a connective tissue has become less popular, and it has now been suggested that the astrocyte plays a role in minutely controlling the neuronal environment by acting as an intermediary between the blood stream and the neural parenchyma. The oligodentrocyte is thought possibly to play a role in the formation and maintenance of the myelin sheath, and perhaps also to be involved in material transport.

The present book emanates from a meeting of researchers held in March 1956, when some of these problems in the morphology and physiology of the neuroglia were discussed. The book, primarily directed to the researcher and the specialist, should especially be recommended to those who are concerned with the physiology of the nerve cell and of the perineuronal environment. Of central interest were the electron-microscopic descriptions of the glial elements, and particularly of the mysterious neuropil or interstitial space of the gray matter, which now for the first time receives elucidation. Of great interest also were the sections relating to the problem of myelin formation and maintenance and the possible relation of the oligodentrocyte to this process.

Biology of Neuroglia represents an excellent treatment of our present understanding of the morphology and function of these much-neglected elements of the nervous system.

—*RONALD E. MYERS, Capt., MC, USA*

ANOMALIES OF INTESTINAL ROTATION AND FIXATION (Including Mesentericoparietal Hernias), by *Roberto L. Estrada*, B. Sc., M. D., C. M., D. Surg. (McGill), F. R. C. S. (C), F. A. C. S. 161 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $6.50.

This monograph represents one of the few reviews on this subject to be printed, as most of the literature consists of isolated case reports. The author reviewed the literature from 1786 through 1956 with the idea of obtaining a clearer understanding of the way abnormalities of intestinal rotation and fixation are produced. An anatomic and embryologic background are developed which will be very valuable to the surgeon, radiologist, and internist.

With the idea of standardizing the nomenclature as an aid for future publications and analysis, the author develops his own classification. He designates the following stages in rotation and fixation at which anomalies may develop: the period of umbilical cord herniation, the stage of deviation, the stage of realignment, the period of reduction

into the abdomen, and the stage of fixation. Properly, he stresses the midgut as the offending organ in all cases.

Based on this review and on 29 of the author's own cases, he further elucidates the symptomatology, diagnostic aspects, and treatment when the surgeon is confronted with an anomaly in the operating room. From a clinical viewpoint, probably the most important sections are on appendicitis in non-rotation, and on mesentericoparietal herniations.

Complete with excellent diagrams x-ray pictures, and bibliography, this volume should do much to clarify in the clinician's mind a generally misunderstood problem.

—*LESTER J. POPE, Capt., MC, USN*

CLINICAL PROCEDURES IN OCCLUSAL REHABILITATION, by *S. Charles Brecker,* D. D. S., F. A. C. D. 326 pages; 936 illustrations on 428 figures. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $16.

The author of this volume presents the subject of occlusal rehabilitation in a revealing manner. To many dentists occlusal rehabilitation has been avoided as a problem area, or a phase of practice too difficult to attempt without participation in postgraduate training and the purchase of complicated equipment. The limitations controlling successful rehabilitation are stressed. The thorough knowledge of what not to treat seems as paramount to success as the ability to treat correctly those cases selected. The required selection of a specific precision articulator is dispensed with as merely a complication to progress. Blind adherence to the use of the hinge axis for patients who for years have been functioning in eccentric positions may invite trouble. Geometric formulas, complicated theories on occlusion, and function of the temporomandibular joint are not essential to the successful restoration of occlusion. About half of the book deals clearly with step-by-step procedures for the preparation of teeth and the laboratory procedures necessary for completion of the case. Full coverage restorations, fixed and removable partial dentures, and internal attachments are precisely described. Emphasis is placed on pictures for each clinical step. The balance of the book is devoted to a practical grouping of patients needing occlusal reconstruction with step-by-step procedures for each group. Limitations of materials are discussed, as are anatomic abnormalities and muscular habits. The book is easy to read and the technics easily followed. It is highly recommended as a valuable addition to the personal libraries of all dentists. —*THEODORE E. FISCHER, Col., USAF (DC)*

VIRAL ENCEPHALITIS, A Symposium, Fifth Annual Scientific Meeting of the Houston Neurological Society, Texas Medical Center, Houston, Tex. Compiled and edited by *William S. Fields* and *Russell J. Blattner.* 225 pages; illustrated. Charles C Thomas, Springfield, Ill., 1958. Price $7.

During the past five years the Houston Neurological Society has sponsored annual symposia, each based on a single broad topic. The

contributors, highly qualified in their specific fields, present facets of these topics from their own points of view. A serious and, in general, successful attempt is made to integrate present-day knowledge and concepts regarding these important topics.

This 225-page, neatly bound, and clearly printed book contains material presented at the March 1, 1957 meeting, devoted to "viral encephalitis." The papers contain much interesting data not generally available in standard textbooks. A reasonable classification is offered and its defects carefully noted. Exceptionally good is a paper on the epidemiology of western equine and St. Louis encephalitis which points up the importance of arthropod transmission and possible wild animal reservoirs in the viral diseases. A superb paper on clinical and laboratory diagnosis contains clear tables delineating and contrasting the important viral affections of the nervous system. A detailed but easily read discussion of postencephalitic sequelae indicates the prognostic significance of a patient's age at onset. Older concepts of a progressive or chronic viral encephalitis are shown to be questionable. A long, almost monographic, and beautifully illustrated paper describes the pathology of viral diseases in man characterized by nuclear inclusions. Including this topic in an otherwise well balanced symposium, without providing a brief account of pathologic changes found in more common varieties of viral central nervous system disease is unusual. Control of neurotropic viral diseases is most successful in rabies and poliomyelitis and recent trends in these are dealt with methodically and well in the final paper.

As in most symposia the discussions following each paper are entertaining and valuable. The principal defect of this and similar works is the lack of an index. The book is fairly inexpensive and in a few hours gives a thoughtful reader a good picture of viral encephalitis as we see it today. It is the best single volume on the subject and is recommended highly to neurologists, internists, pediatricians, and general practitioners.

—*HENRY W. HOGAN, Maj., MC, USA*

OCULAR ALLERGY, by *Frederick H. Theodore,* M. D., and *Abraham Schlossman,* M. D., with chapters by *William B. Sherman,* M. D., and *Robert S. Coles,* M. D. 420 pages; illustrated. The Williams & Wilkins Company, Baltimore, Md., 1958. Price $12.

This is the first recent book on ocular allergy, and the need for one has been evident for many years. Ocular allergy forms an increasing part of ophthalmologic practice. Many allergies are not recognized as such, while others are erroneously so diagnosed.

The authors have thoroughly covered those conditions known to be allergic in origin, and have called attention to many conditions that could be possibly explained by hypersensitivity. The text is a practical one, and those interested in allergy will enjoy reading it. The book is well written; it has a good index and bibliography and few typographical errors. —*NELSE O. OLSON, Capt., MC, USN*

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A GUIDE TO HUMAN PARASITOLOGY FOR MEDICAL PRACTITIONERS (*Blacklock* and *Southwell*), revised by *T. H. Davey*, O. B. E., M. D., D. T. M. 6th edition. 222 pages; with three colored plates and 119 illustrations in the text. The Williams and Wilkins Co., Baltimore, Md., 1958. Price $7.00.

This book is a guide for clinicians who do not have the assistance of a clinical laboratory and must depend upon their own examination of samples for a laboratory diagnosis. It is assumed that the reader has had no experience in the field and the text is designed to develop proficiency in the diagnosis of parasitic infections.

An introduction to the use of the microscope and the preparation and examination of samples is followed by a discussion of the various classes of parasites. The habitat, morphological characters, life history, pathogenicity, diagnosis, and prevention are presented in a concise manner for each parasite. There are numerous keys which aid in identification, and the life histories are diagrammatically presented in a simplified manner. Further identification of parasites is aided by the 119 illustrations. Although this text was written for the clinician, it may be used by anyone interested in attaining proficiency in identification of human parasites. —*JOHN E. STAUCH, Capt., USAF (MSC)*

MODERN TRENDS IN ENDOCRINOLOGY, Edited by *H. Gardiner-Hill*, M. D., F. R. C. P. 311 pages; illustrated. Paul B. Hoeber, Inc., medical book department of Harper & Brothers, New York, N. Y., 1958. Price $13.50.

This volume, which consists of 21 chapters, lives up to its title admirably. It covers most of the major advances in endocrinology today, and indicates the likely lines of future progress. The articles are for the most part informative and concise with an excellent up-to-date bibliography. The majority of the authors are well established British investigators, including Pitt-Rivers, Randall, McCance, Black, Prunty, and Cope. This reviewer and Professor Swan of the Mayo Clinic were the only American authors in the volume.

As might be expected, the subject matter is quite varied, ranging from the theoretical aspects of the significance of the stress concept in clinical medicine, and hormonal factors in breast development and milk secretion, to the very practical problems of uses of steroids in the treatment of rheumatic and allergic disorders, and endocrine therapy of malignancy of the breast and prostate. Particularly outstanding in this volume is the article by Dr. Randall, entitled "Endocrine Factors in the Syndrome of Diabetes Mellitus," which represents the most concise summation of the extensive investigations done by this investigator and Professor Frank Young that has yet appeared in print.

The article by Gilliland on the etiology and treatment of exophthalmos, while quite adequate, could possibly have been improved by reference to recent advances in hypophysectomy and stalk transection in the treatment of this condition. Similarly, the article concerned with tests of thyroid function did not have a discussion of a butanol extractable

iodine, and might have been of more benefit to many physicians had it included a brief discussion of the current controversy concerning hypometabolism without hypothyroidism. Neither of these chapters referred specifically to the use of thyroid-stimulating hormone or prolonged treatment with thyroid extract in the diagnosis of hyperthyroidism, especially when associated with the problem of exophthalmos without other evidence of increased thyroid activity.

It is a primary datum of observation that one does not criticize a work for not doing what it never intended to do. Obviously the whole field of endocrinology could not be covered in such a volume and one hopes that the reception of this work will be sufficient to stimulate further issues in this much needed series. There is no question, however, that the subject matter presented in this volume is as broad a coverage of the field as could be done in the space allotted.

This book should be in every medical library and will undoubtedly be of considerable value to endocrinologists as well as those internists and gynecologists with an interest in this subject. The print is legible and large, and the changing styles and balance of the subject matter is quite refreshing and makes for enjoyable reading. I believe that the distinguishing quality of this volume, however, is that it is more up-to-date than many current periodicals. In addition, it provides a very refreshing insight into authoritative British thinking on a variety of important subjects.—*PAUL J. ROSCH, Capt., MC, USA*

BONE AND RADIOSTRONTIUM, by *Arne Engstrom, Rolf Bjornerstedt, Carl-Johan Clemedson,* and *Arne Nelson.* 139 pages; illustrated. John Wiley and Sons, Inc., New York, N. Y., 1958. Price $8.75.

This short monograph consists of a review of experimental work performed by the authors on the metabolism of radiostrontium, the physiology and micromorphology of skeletal tissue, and the development of a dosimetric model based on these studies for an estimation of the maximum permissible dose of Strontium^{90}.

The introductory chapter is concerned with metabolism of strontium in the mammalian organism, well documented with 70 references, many of them appearing in the American literature. The language is not always fluent and a few typographical or translator's errors occur.

Three chapters discuss the distribution and ultrastructure of bone salts, and the distribution of Sr^{90} in the skeleton. The technics used in this study, x-ray microscopy or historadiography, micro-interferometry, and autoradiography, are adequately discussed and described. The illustration quality is good, though the lack of labelled anatomic landmarks may make orientation difficult.

Theorectical calculation of the dose rate distribution from Beta emitters comprises a whole chapter. One wonders whether such a sophisticated development of dosimetry (30 pages) is warranted, as the authors have chosen as their model for spongiose bone an "infinite sandwich"

of alternate layers of radioactive and non-radioactive material; this model, according to their admission, is only approximate.

An attempt at estimation of the maximum permissible body burden of Strontium^{90} is made in the last chapter, and is actually based on a system of dosimetry commonly used, rather than on the system developed by the authors. As a first approximation the dose to bone is calculated to be 5.8 mrem per day per gram of bone, which estimate, assuming Sr^{90} is concentrated homogeneously in the osseous portion of bone, is raised to 25 mrem per day. Taking into account the deposition of Sr^{90} in "hot spots," where the maximal dose rate may be 10 times higher, estimates of local dose rates in bone of from 300 to 500 mrem per day per gram of bone are made by the authors. This is from a total body burden of 1 uc of Sr^{90}, the current maximum permissible body burden. On the basis of their studies the authors conclude that a body burden of less than 0.1 uc Sr^{90} is tolerable.

The experimental data is well presented and the technics described will doubtless be of value to other investigators. Final development of an estimate for the maximum permissible body burden of Sr^{90} is not as rigorous as the material which precedes it, and will probably be subject to critical review.—*THOMAS G. MITCHELL, Lt., MSC, USN*

CARDIOVASCULAR COLLAPSE IN THE OPERATING ROOM, by *Herbert E. Natof*, M. D. and *Max S. Sadove*, M. D. Foreword by *Warren H. Cole*, M. D. 197 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1958. Price $6.

This volume encompasses the inclusive field of cardiovascular collapse as differentiated from the more dramatic and less precise term, cardiac arrest, in an effort to correlate the numerous causes and effects of the physiologic imbalances resulting in operating room emergencies. It recognizes that there is now no uniform method for classifying data since the criteria for classification and interpretation vary so greatly. Emphasis is placed on the multiple predisposing and precipitating factors involved. Utmost stress is made on an adequate preoperative evaluation of the patient's condition and the correction of basic pathophysiologic mechanisms. Only full co-operation of the entire medical staff can fully assess the patient as a surgical risk and institute corrective measures for existing deficiencies. The prophylactic measures begun with the first physician examining the patient must be carried through by an alert and co-operative surgical team where free communication exists.

The book is well organized and logically presented. The inadequacies of the present reporting methods are noted and 775 cases are analyzed to fully implicate the multiple factors involved. Causative mechanisms are discussed individually and completely. These include the management of preexisting cardiovascular and pulmonary diseases, hypovolemia and anemia, various endocrine abnormalities and the special problems encountered with the extremes of age, obesity, drunkenness, racial variations and specific diseases. The blood coagulation disturb-

ances and transfusion reactions are reviewed and diagnostic and therapeutic measures are suggested. Thirty-three cases are presented by protocol and primary causes of the complications are indicated. Prevention of catastrophe depends on an awareness of special problems and the institution of the necessary precautions. The anesthesiologist's perception of early signs of impending danger are of greatest importance. Constant watchfulness is the keynote in successful diagnosis and treatment and this cannot be replaced by monitoring devices. Treatment is divided into specific and nonspecific elements and a complete emergency treatment-cart is described.

The tables are easily read and the figures are accomplished with clarity. The bibliography is extensive and the index is complete. This book is recommended not only for anesthesiologists but for all physicians who wish to guide a patient successfully through a surgical procedure. — *ROBERT E. LAU, Col., USAF (MC)*

CLINICAL ORTHOPAEDICS, by *Anthony F. DePalma,* editor-in-chief, with the assistance of the Associate Editors, The Board of Advisory Editors, The Board of Corresponding Editors. No. 10, Fall, 1957. 367 pages; illustrated. J. B. Lippincott Company, Philadelphia, Pa., 1957. Price $7.50.

Volume 10 of Clinical Orthopaedics is dedicated to Phemister of Chicago, a great general surgeon who, based on his experience in Germany, has probably contributed more to clinical application of bone pathology and bone physiology than any single man in our century. The volume contains four sections dealing with the affections of growth centers, the part of basic physiology of metabolic bone disorders which could not be included in Volume 9, general orthopedics and the fourth section entitled "Items"—a total of 30 articles. The editor has again shown his ability in presenting broad aspects of orthopedics and borderline fields.

As to the practical value contained in the individual papers, McElvenny's discussion of the treatment of hip fractures is probably the most outstanding. The general attitude toward that problem is one of resignation to the necessity of accepting a high percentage of undesirable results. McElvenny's discussion, based upon clear indications, meticulous technic and 20 years of experience, shows ways of avoiding disappointments which have been entirely successful in his hands. Of great value to the orthopedist also is Garland's article on long-range effects of radiation on bone, written with great clarity and authority. Martin and Pipkin's article on treatment of avulsion of the ischial tuberosity has beautiful sketches illustrating the mechanism of injury. The original mode of presentation certainly enhances the teaching value of the paper. Calvo presents a nice piece of research on the growth of the female adolescent spine which indicates that the criteria generally used at present are not entirely reliable. Katz presents an end-result study of 111 cases of Legg-Perthes disease treated by various forms of non-weightbearing on the affected side. His conclusions

are extremely cautious, and should be, because the results shown fall short of the ones seen in entirely untreated cases.

One wonders whether the importance of the acetabular index is not over-emphasized as it does not seem to have any bearing on the function or on the development of later complications. It is interesting to note the great variations in the present attitude toward credit given to previous writers on the subject. Thus, Howorth devotes almost three pages to the history of the slipping of the upper femoral epiphysis while J. E. M. Thomson revives turnbuckle correction in fresh fractures, apparently unaware of the original work of Fabricius Hildanus in 1629 and the extensive use of the method of Hackenbruch in Germany during World WarII. The volume again is of great interest and value to the orthopedist, and, beyond that, to anyone interested in conditions affecting the skeletal system.—*ERNST DEHNE, Col., MC, USA*

PHARMACOLOGY IN MEDICINE, A Collaborative Textbook, edited by *Victor A. Drill,* Ph. D., M. D. 2d edition. 1,273 pages; illustrated. Published by McGraw-Hill Book Co., Inc., Blakiston Division, New York, N. Y., 1958. Price $19.50.

This comprehensive volume of multiple authorship is intended as a textbook and reference, a purpose generally well fulfilled. The material is well arranged, and major headings divide the chapters into drug usages, organ systems, and specific therapeutic agents. The chief advantage of its multiple authorship is the presentation of authoritative information and opinions on a myriad of subjects. Individual drugs are discussed from the standpoint of mechanism of action, effect in the body, therapeutic action, and available forms. The discussions of physiology and pharmacology are written simply and understandably, with considerable emphasis placed on drug action and least emphasis on the clinical aspects.

This book is well written, fulfills a definite need as a pharmacology text, and is a useful reference for students and practitioners. Its chief deficiency from the clinician's standpoint—and this is not too serious—is its brevity regarding actual drug dosage and clinical application.—*R. J. PEARSON, Jr., Capt., MC, USN*

CARBON DIOXIDE THERAPY, A Neurophysiological Treatment of Nervous Disorders, edited by *L. J. Meduna,* M. D. 2d edition. 541 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $14.50.

This volume, sponsored by the Carbon Dioxide Research Association, merits attention as a handbook on the use and present status of carbon dioxide in the treatment of nervous disorders. The several tested technics of carbon dioxide therapy, the indications and contraindications of their use, and the nature and extent of the clinical results achieved, are discussed in great detail. The collected papers also provide a summary of the available information and emergent understandings relative to the physiologic basis of this form of treatment. In addition to its importance to the psychiatrist, this symposium

can be expected to be of great interest to the general practitioner and to research workers concerned with the physiologic changes associated with emotional stress response.

Separate papers are devoted to the use of carbon dioxide therapy in anxiety reactions, anxiety states and phobic reactions, pediatrics, obsessive-convulsive neuroses, depressive reaction, sex deviations, and obstetrics. Exclusive of the favorable response of the last two categories of patients, it appears that clinical recovery or very substantial improvement was observed in approximately 70 per cent of the combined 318 patients who had received appropriate carbon dioxide therapy. Analagous data pertain to a statistical approach to the study of 656 other patients, 68 per cent of whom showed improvement as a result of carbon dioxide treatment. Judged in terms of the Jackman Neurotic Index, about 62 per cent with anxiety states (370 patients) showed clinical recovery, while an additional 19 per cent were greatly improved, following carbon dioxide therapy. Data derived from the use of the Minnesota Multiphasic Personality Inventory (MMPI) support the thesis that patients initially resistant to psychotherapy, tend to become more responsive when treated with carbon dioxide, plus psychotherapy. —*THADDEUS J. DOMANSKI, Col., USAF (MSC)*

THE HANDICAPPED AND THEIR REHABILITATION, edited by *Harry A. Pattison,* M. D., F. A. C. P. Introduction by Major General *Melvin J. Maas,* USMCR Ret. 944 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1957. Price $14.75..

This book is written by 44 coauthors, all experts in their various fields of rehabilitation. It is divided into four parts: the foundations of rehabilitation, the disabilities, the rehabilitation team, and special problems. The concept of rehabilitation is described by one of the authors as "a system of caring for the patient from his bed of acute illness to his return to the community once again independent and capable of assuming his responsibility as a citizen. It has its origin in the belief that the care of a patient does not end with the subsiding of an acute illness, but that it extends beyond that point to restoration of individuality, personality, and human dignity." Rehabilitation applies to all patients, not only the severely disabled, and in this sense is the prime responsibility of all physicians. However, the full rehabilitation of the patient is not possible without the skills of the various members of the team whose services must be co-ordinated and integrated. In addition to the physician, the team may be made up of a rehabilitation co-ordinator; physical therapist; occupational therapist; rehabilitation nurse; medical social worker; vocational counselor; clinical psychologist; and corrective, educational, and manual arts therapists. These team members have contributed individual chapters describing the specific role and function of each.

The book is of value to physicians in general, and to all members of the allied co-professional groups.

—*ANIELLO MASTELLONE, Lt. Col., MC, USA*

THE IMPACT OF THE ANTIBIOTICS ON MEDICINE AND SOCIETY, Monograph II, *Institute of Social and Historical Medicine, The New York Academy of Medicine.* Edited by *Iago Galdston,* M. D. 222 pages. International Universities Press, Inc., New York, N. Y., 1958. Price $5.

This book is composed of several chapters, each dealing with some phase of antibiotics and is a compilation of papers given at the Institute of Social and Historical Medicine of the New York Academy of Medicine. The historical development of these drugs has been covered in such chapters as "The Microbiology of the Soil and the Antibiotics," and "The Role of Industry in the Mass Production of Antibiotics." It covers, in general fashion, the effects of these drugs in our present society as they influence infectious diseases. The book is of interest for those who desire a general historical review of the development of the antibiotics and their present status in our society.

DOSS O. LYNN, Col., MC, USA

DIAGNOSTIC MEDICAL PARASITOLOGY, by *Edward K. Markell,* Ph. D., M. D., and *Marietta Voge,* M. A., Ph. D. 276 pages; 115 figures, including 5 in color. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $7.

Markell and Voge wrote this book to present in readily available form the essential facts needed by the medical student and clinical laboratory technologist for the diagnosis of human parasitic disease. In this venture they have succeeded admirably.

The book is well organized and written in a clear and simple manner. The 115 figures found in its 276 pages contribute significantly to the usefulness of the text. They have obviously been carefully chosen for their practical diagnostic utility rather than for mere encyclopedic coverage of the subject. The initial two chapters state the purpose and frame of reference of the book, and provide a lucid discussion of the concept of parasitism. Succeeding chapters group the various parasites according to their organ system of predilection, rather than following the classical phylogenetic format. This parallels the approach of the physician examining a patient—he first considers the symptomatology and organ systems involved; identification of the etiologic agent comes later. The short chapter on pseudoparasites and pitfalls will alert the unwary and assist those for whom parasitologic diagnosis is not a primary occupation. Special methods, procedures, and materials are included in a form well adapted for ready reference. The treatment of parasitic disease has been purposely omitted since therapy is changing too rapidly for a book to remain current.

Although *Diagnostic Medical Parasitology* was written primarily as a textbook, military physicians and laboratory personnel will find it valuable as a practical, ready reference manual, to be used in conjunction with the more extensive texts on clinical parasitology and tropical medicine. —*HERSCHEL E. GRIFFIN, Lt. Col., MC, USA*

TEXTBOOK OF OBSTETRICS AND OBSTETRIC NURSING, by *Mae M. Bookmiller*, R. N., and *George Loveridge Bowen*, M. D., with a section on the newborn by *Harry Bakwin*, M. D. 725 pages; illustrated. With original drawings by *Frank Netter*, B. S., M. D. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $6.

This textbook is a concise yet encyclopedic exposition of the currently accepted theory and practice of obstetrics and is particularly directed to the student and practicing nurse. It is well organized, written, and illustrated. The unit "Nursing in the Neonatal Period" is a particularly valuable addition to the book. It covers the subject very well without undue verbosity. The index is very adequate. A glossary of words and terms pertaining to the subjects covered is a helpful and handy addition not often found in texts of this character.

A list of examination questions at the end of each chapter offers an excellent means of quick review of the subject matter covered. The illustrations are profuse, clear and to the point. All-in-all the book should prove a great practical value to student, teacher and practicing nurse.—*ROY E. CROWDER, Capt., MC, USN*

AN INTRODUCTION TO EXPERIMENTAL SURGICAL STUDIES, by *W. J. Dempster*, F. R. C. S. 463 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $10.

This book is intended primarily for the postgraduate surgical student engaging in experimental surgery. Any surgeon interested in understanding the relationship between clinical and experimental surgery would greatly benefit by it. The great advances in modern surgery are the direct result of applied physiologic principles and experimentation. The text is easy to read, entertaining, and matter-of-fact, so typical of the British author. It is well indexed and illustrated. The bibliography is copious, comprehensive, and is conveniently located at the end of each topic discussed. The book is divided into an introduction and eight chapters, and throughout the relationship of clinical and experimental surgery is well correlated, providing a wide range of references and critical commentary which serve as a springboard to one interested in experimental surgery. The editor is to be congratulated on the concise and lucid manner in which he reveals the close relationship between clinical and experimental surgery.

—*OAKLEY K. PARK, Lt. Col., USAF (MC)*

INTESTINAL OBSTRUCTION, by *Claude E. Welch*, M. D., D. Sci. (Hon.) Illustrated by *Muriel McLatchie Miller*. 376 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $10.50.

The organization of this volume into general considerations (anatomy, diagnosis, and etiology), fundamental aspects of therapy (time of operation, intubation, and replacement therapy), and types of obstruction is fully appreciated when used for ready reference. Presentation style and descriptive terms used result in easy and interesting reading, without the usual excess of tables and graphs. The precise manner is uncluttered with multiple variations or repetitive terms.

Chapters on tubing and postoperative obstruction are as informative as any this reviewer has read. Treatment is presented in a logical and physiologic manner, as each type of obstruction is discussed.

Applying the essentials of this text to clinical surgery will lead to improved results in diagnosis and treatment of intestinal obstruction. While not a medical student's book, it is highly recommended for residents and practicing surgeons.

—VICTOR C. STRATTON, Capt., MC, USN

DYNAMICS OF PSYCHOTHERAPY, The Psychology of Personality Change, by *Percival M. Symonds,* Ph. D. Volume III, Procedures. 634 pages. Grune and Stratton, Inc., New York, N. Y., 1958. Price $6.50.

This third volume of *Dynamics of Psychotherapy* is concerned with the role of the therapist, his responsibilities, goals, and technics. Symonds regards interpretation as the chief function of the therapist in psychotherapy, and this interesting and controversial issue is the topic of 10 of the 19 chapters. When to intervene, when to clarify, when to question, when to keep silent—all are considered. What patient resistance means and how it should be handled, how transference is managed, and how the patient's anxiety is related to the therapist's interpretations are also detailed. The therapist's use of reassurance (and all therapists use it), suggestion, commands and advice, is explained.

This volume should be of use to the ordinary practitioner who is interested in psychotherapeutic aspects of medicine, and to the beginning psychiatrist who would like a systematic textbook which will tell him not only what to do, but why to do it.

—WALTER L. WILKINS, Comdr., MSC, USNR

RECONSTRUCTIVE AND REPARATIVE SURGERY, by *Hans May,* M. D., F. A. C. S. 2d edition. 1,115 pages; with 1,030 illustrations, 55 in color. F. A. Davis Company, Philadelphia, Pa., 1958.

This is an excellent, well-organized textbook on plastic surgery. It is divided into five sections and each section contains an excellent bibliography with ample illustrations.

The first section deals with general principles of plastic surgery with a particularly good portion devoted to a discussion of grafts. The next three sections are appropriately divided and refer to the respective regions of the body. The fifth section contains a group of illustrated cases which makes interesting reading. The present edition is the first revision in 10 years, and most of the changes concern the treatment of burns, use of antibiotics, and modern concepts of transplantation.

The book has much useful material and is presented in a clear, concise manner. General surgeons should find it a helpful and valuable text to have on hand, especially when the services of a qualified plastic surgeon are not readily available.

—FERDINAND V. BERLEY, Capt., MC, USN

New Books Received

Books received by the *U. S. Armed Forces Medical Journal* are acknowledged in this department. Those of greatest interest will be selected for review in a later issue.

PRINCIPLES OF INTERNAL MEDICINE. Editors: *T. R. Harrison, Raymond D. Adams, Ivan L. Bennett, Jr., William H. Resnik, George W. Thorn,* and *M. M. Wintrobe.* 3d edition. 1,839 pages; illustrated. The Blakiston Division, McGraw-Hill Book Company, Inc., New York, N. Y., 1958. Price $18.50.

THE ACUTE ABDOMEN, by *William Requarth,* M. D. Foreword by *Warren H. Cole,* M. D. 2d edition. 313 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $6.50.

A TEXT ON SYSTEMIC PATHOLOGY, Volume I, edited by *Otto Saphir,* M. D. 865 pages; with over 800 illustrations. Grune and Stratton, Inc., New York, N. Y., 1958. Price $32.

A PHYSICIAN LOOKS AT PSYCHIATRY, by *Jacques M. May,* M. D. 189 pages. The John Day Company, New York, N. Y., 1958. Price $3.50.

PATHOLOGY FOR THE PHYSICIAN, by *William Boyd,* M. D., Dipl. Psychiat., M. R. C. P. (Edin.), Hon. F. R. C. P. (Edin.), F. R. C. P. (Lond.), F. R. C. S. (Can.), F. R. S. (Can.), LL. D. (Sask), (Queen's), D. Sc. (Man.), M. D. (Oslo), 6th edition, thoroughly revised. 900 pages; 489 illustrations and 12 plates in color. Lea & Febiger, Philadelphia, Pa., 1958. Price $17.50.

THE DOCTOR IN PERSONAL INJURY CASES, by *Harold A. Liebenson.* Foreword by *Sidney S. Greenspahn,* M. D. 123 pages. Published by The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $4.

THE PSYCHOLOGY OF MEDICAL PRACTICE, by *Marc H. Hollender,* M. D. 276 pages. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $6.50.

MILESTONES IN MODERN SURGERY, by *Alfred Hurwitz,* M. D., and *George A. Degenshein,* M. D., with a foreword by *J. Englebert Dunphy,* M. D. 520 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Department of Harper & Bros. New York, N. Y., 1958. Price $15.

A MANUAL ON CARDIAC RESUSCITATION, by *Robert M. Hosler,* M. D., F. A. C. S. 2d edition. 208 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $5.50.

VARIABLES RELATED TO HUMAN BREAST CANCER, by *V. Elving Anderson, Harold O. Goodman,* and *Sheldon C. Reed.* A Study from the Dight Institute of Human Genetics. 172 pages; illustrated. The University of Minnesota Press, Minneapolis, Minn., 1958. Price $4.

CLINICAL STUDIES IN CULTURE CONFLICT, edited by *Georgene Seward,* Ph. D. 598 pages; illustrated. The Ronald Press Company, New York, N. Y., 1958. Price $7.

ACTION OF RADIATION ON TISSUES, An Introduction to Radiotherapy, by *A. Lacassagne* and *G. Gricouroff.* Translated by *Clarence C. Lushbaugh,* M. D., and *Gretchen R. Riese,* M. S. 199 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $6.25.

INDUSTRY AND TROPICAL HEALTH III, Proceedings of the Third Conference of the Industrial Council for Tropical Health at The Harvard School of Public Health, Boston, Mass., 16-18 April 1957. Published by The Harvard School of Public Health for The Industrial Council for Tropical Health, Boston, Mass., 1957. 261 pages; illustrated. Price $10. (professional rate $5.)

20TH CENTURY CHEMISTRY, by *Joseph I. Routh,* Ph. D. 2d edition. 613 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $6.50.

CARBON DIOXIDE THERAPY, A Neurophysiological Treatment of Nervous Disorders, edited by *L. J. Meduna,* M. D. 2d edition. 541 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $14.50.

ATRIAL ARRHYTHMIAS, DIGITALIS AND POTASSIUM, by *Bernard Lown,* M. D., and *Harold D. Levine,* M. D. 222 pages; illustrated. Landsberger Medical Books, Inc., New York, N. Y. 1958. Price $6.90.

MEDICAL ELECTRICAL EQUIPMENT, Principles, Installation, Operation and Maintenance of Electrical Equipment used in Hospitals and Clinics. Advisory Editor: *Robert E. Molloy,* M. B., F. F. A., R. C. S. 21 Contributors. 312 pages; illustrated. Philosophical Library, Inc., New York, N. Y.. 1958. Price $15.

FORENSIC MEDICINE, by *Keith Simpson,* M. D. (Lond.) 352 pages; illustrated. The Williams & Wilkins Company, Baltimore, Md., 1958. Price $7.

THE MEDICAL ASSISTANT, A Guidebook for the Nurse, Secretary, and Technician in the Doctor's Office, by *Miriam Bredow.* 430 pages; illustrated. The Blakiston Division, McGraw-Hill Book Company, Inc., New York, N. Y., 1958. Price $7.50.

PEDIATRIC SURGERY, by *Orvar Swenson,* M. D., F. A. C. S. Foreword by *William E. Ladd,* M. D. 750 pages; 980 illustrations. Appleton-Century-Crofts, Inc., New York, N. Y., 1958. Price $20.

MODERN CLINICAL PSYCHIATRY, by *Arthur P. Noyes,* M. D., and *Lawrence C. Kolb,* M. D. 5th edition. 694 pages. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $8.

NUTRITION FOR PRACTICAL NURSES, by *Phyllis S. Howe,* B. S. 2d edition. 219 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $2.75.

PROGRESS IN PSYCHOTHERAPY, Volume III. TECHNIQUES OF PSYCHOTHERAPY, edited by *Jules H. Masserman,* M. D., and *J. L. Moreno,* M. D. 324 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $8.50.

SURGERY IN INFANCY AND CHILDHOOD, A Handbook for Medical Students and General Practitioners, by *Matthew White,* M. A., M. B., Ch. B., F. R. F. P. S. (Glas.), F. R. C. S. (Edin.), and *Wallace M. Dennison,* M. D., F. R. F. P. S. (Glas.), F. R. C. S. (Edin.), F. I. C. S. 444 pages; illustrated. The Williams & Wilkins Company, Baltimore, Md., 1958. Price $9.50.

THE YEAR BOOK OF ENDOCRINOLOGY (1957-1958 Year Book Series), edited by *Gilbert S. Gordan,* M. D., Ph. D., F. A. C. P. 381 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $7.50.

THE YEAR BOOK OF PATHOLOGY AND CLINICAL PATHOLOGY (1957-1958 Year Book Series), edited by *William B. Wartman,* B. S., M. D. 478 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $8.

HUMAN BLOOD IN NEW YORK CITY, A Study of Its Procurement, Distribution and Utilization. Conducted by *The Committee on Public Health, the New York Academy of Medicine,* under the direction of its *Subcommittee on Blood Survey. H. D. Kruse,* M. D., General Director of Study. 147 pages; illustrated. The New York Academy of Medicine, New York, N. Y., 1958.

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, Volume 70, Art. 3, Pages 277-762, June 3, 1958. Editor in Chief, *Otto V. St. Whitelock;* Managing Editor, *Franklin N. Furness;* Associate Editor, *Peter A. Sturgeon.* "Animal Disease and Human Health." Conference Chairman and Consulting Editor, *James Lieberman.* 485 pages; illustrated. New York Academy of Sciences, New York, N. Y., 1958.

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THE ANALYSIS OF DREAMS, by *Medard Boss,* M. D. Translated by *Arnold J. Pomerans.* 223 pages. Philosophical Library, Inc., New York, N. Y., 1958. Price $6.

JUVENILE DELINQUENCY, edited by *Joseph S. Roucek,* with the collaboration of 14 eminent authorities. 370 pages. Philosophical Library, Inc., New York, N. Y., 1958. Price $10.

DENTAL CLINICS OF NORTH AMERICA, Symposium on The Interrelationship of Oral and Systemic Disease. *Lester W. Burket,* D. D. S., M. D., Consulting Editor. 543 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION, Volume 49, 1957, Published June, 1958. Editorial staff: *Carl M. Gambill,* A. B., M. D., M. P. H.; *James R. Eckman,* A. B., M. A., Ph. D.; *M. Katharine Smith,* B. A.; *Florence L. Schmidt,* B. S. E.; *George G. Stilwell,* A. B., M. D.; *Guy Whitehead,* B. A., M. A., Ph. D. Senior Consultants: *Richard M. Hewitt,* B. A., M. A., M. D., and *John R. Miner,* B. A., Sc. D. Compiled by *George G. Stilwell,* A. B., M. D. 827 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $13.

METHODS IN PHYSICAL EDUCATION, An Illustrated Textbook for Students Preparing to Teach Boys and Girls in Secondary Schools, by *Hilda Clute Kozman,* Ph. D.; *Rosalind Cassidy,* Ed. D.; and *Chester O. Jackson,* Ed. D. 3d edition. 549 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $6.

TEXTBOOK OF BIOCHEMISTRY, by *Benjamin Harrow*, Ph. D., and *Abraham Mazur*, Ph. D. 7th edition. 557 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $7.50.

DISEASES OF THE ESOPHAGUS, by *J. Terracol* and *Richard H. Sweet*. 682 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $20.

PYE'S SURGICAL HANDICRAFT, A Manual of Surgical Principles, Minor Surgery, and other matters connected with the work of surgical dressers, house surgeons and practitioners, edited by *Hamilton Bailey*, F. R. C. S. (Eng.), F. A. C. S., F. R. S. (Edin.). 17th edition, fully revised. 802 pages; with 860 illustrations. The Williams & Wilkins Company, Baltimore, Md., 1956. Price $10.

PSYCHIATRIC RESEARCH REPORTS OF THE AMERICAN PSYCHIATRIC ASSOCIATION, edited by Members of the Committee on Research, 1956-1957: *Nathan S. Kline*, M. D., Chairman; *Robert A. Cleghorn*, M. D., *Milton Greenblatt*, M. D., *Harold E. Himwich*, M. D., *William T. Lhamon*, M. D., *Benjamin Pasamanick*, M. D. No. 9; "Research in Psychiatry with Special Reference to Drug Therapy." Papers presented at the Regional Research Conference held under the joint auspices of the American Psychiatry Association and the Medical Schools and Affiliated Hospitals of Philadelphia, Pa., 16-17 Nov. 1956. 181 pages; illustrated. American Psychiatric Association, Washington, D. C., 1958. Price $2.

ELECTROCARDIOGRAM CLINICS, by *Joseph E. F. Riseman*, M. D., and *Elliot L. Sagall*, M. D. Clinic I: The Diagnosis and Treatment of Angina Pectoris; Clinic II: The Role of the Electrocardiogram in Establishing the Diagnosis of Acute Myocardial Infarction; Clinic III: Problems in the Diagnosis of Acute Myocardial Infarction; Clinic IV: The Role of the Electrocardiogram in the Diagnosis of Pulmonary Embolism. 259 pages; illustrated. The Macmillan Company, New York, N. Y., 1958. Price $10.50.

PSYCHOPHARMACOLOGY, Pharmacologic Effects on Behavior, edited by *Harry H. Pennes*, M. D., D. Med. Sc. (Neurology). With 41 participants. 362 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, N. Y., 1958. Price $8.

ESSENTIALS OF GYNECOLOGY, by *E. Stewart Taylor*, M. D. 502 pages; 343 illustrations, 4 in color. Lea & Febiger, Philadelphia, Pa., 1958. Price $12.

MANUAL OF MEDICAL EMERGENCIES, by *Stuart C. Cullen*, M. D., and *E. G. Gross*, M. D. 3d edition. 302 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $5.75.

PANIC AND MORALE, Conference Transactions. *The New York Academy of Medicine* and *The Josiah Macy Jr. Foundation*. Edited by *Iago Galdston*, M. D. *Hans Zetterberg*, Ph. D., Research Associate. 340 pages. International Universities Press, Inc., New York, N. Y., 1958. Price $5.

OPHTHALMIC PLASTIC SURGERY, by *Sidney A. Fox*, M. S. (Ophth.), M. D., F. A. C. S. 2d revised edition. 324 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $15.

ORR'S OPERATIONS OF GENERAL SURGERY, by *George A. Higgins*, M. D., F. A. C. S., and *Thomas G. Orr*, Jr., M. D., F. A. C. S. 3d edition. 1,016 pages; with 1,990 step-by-step illustrations on 835 figures. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $20.

CLINICAL ENZYMOLOGY, edited by *Gustav J. Martin,* Sc. D. 241 pages; illustrated. Little, Brown and Company, Boston Mass., 1958. Price $6.

BONE AND RADIOSTRONTIUM, by *Arne Engstrom, Rolf Bjornerstedt, Carl-Johan Clemedson,* and *Arne Nelson.* 139 pages; illustrated. John Wiley and Sons, Inc., New York, N. Y., 1958. Price $8.75.

REGIONAL ILEITIS, by *Burrill B. Crohn,* M. D., and *Harry Yarnis,* M. D., with special contributions by *Richard H. Marshak,* M. D., and *David A. Turner,* Ph. D. 2d revised edition. 239 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $7.25.

PEDIATRIC GYNECOLOGY, With Sections on Urology and Proctology, by *Goodrich C. Schauffler,* M. D. 4th edition. 349 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $9.

PRIVATE, by *Lester Atwell.* 499 pages. Simon and Schuster, New York, N. Y., 1958. Price $5.

ANATOMY AND PHYSIOLOGY LABORATORY MANUAL, by *Arthur W. Glass,* M. A., Ph. D., and *Charles L. Hamrum,* M. S., Ph. D. 2d edition. 95 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $1.75.

FUNDAMENTALS OF CHEMISTRY, by *L. Jean Bogert,* Ph. D. 8th edition. 615 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $5.50.

MODERN TRENDS IN ENDOCRINOLOGY, edited by *H. Gardiner-Hill,* M. D., F. R. C. P. 311 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Department of Harper and Brothers, New York, N. Y., 1958. Price $13.50.

TEXTBOOK OF OBSTETRICS AND OBSTETRIC NURSING, by *Mae M. Bookmiller,* R. N., and *George Loveridge Bowen,* M. D., with a section on the newborn by *Harry Bakwin,* M. D. 725 pages; illustrated with original drawings by *Frank Netter,* M. D. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $6.

THE GUINEA PIG IN RESEARCH, Biology—Nutrition—Physiology, by *Mary Elizabeth Reid,* Ph. D. 87 pages; 7 halftone plates, 12 tables, bibliography of 227 items. Human Factors Research Bureau, Inc., National Press Building, Washington, D. C. Price $2.

ORTHOPEDIC DISEASES, Physiology—Pathology—Radiology, by *Ernest Aegerter,* M. D., and *John A. Kirkpatrick,* Jr., M. D. 602 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $12.50.

TEXTBOOK OF ORGANIC CHEMISTRY, by *Carl R. Noller.* 2d edition. 654 pages; illustrated. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $7.

GOODNOW'S HISTORY OF NURSING, by *Josephine A. Dolan,* R. N., M. S. 10th edition. 422 pages; illustrated with 255 figures. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $5.

RELIGIOUS DOCTRINE AND MEDICAL PRACTICE, by *Richard Thomas Barton,* M. B., B. S., M. D., F. A. C. S. Foreword by *Raymond B. Allen,* M. D. 94 pages. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $3.75.

ELECTRONIC INSTRUMENTATION FOR THE BEHAVIORAL SCIENCES, by *Clinton C. Brown,* Ph. D., and *Rayford T. Saucer,* Ph. D. A Monograph in The Bannerstone Division of American Lectures in Objective Psychiatry, edited by *W. Horsley Gantt,* M. D. 160 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $5.50.

THE EFFECTS OF A THREATENING RUMOR ON A DISASTER-STRICKEN COMMUNITY, by *Elliott R. Danzig, Paul W. Thayer,* and *Lila R. Galanter.* Disaster Study No. 10, Disaster Research Group, Division of Anthropology and Psychology. Prepared for Federal Civil Defense Administration and The Disaster Research Group (Formerly The Committee on Disaster Studies). 116 pages; illustrated. Publication 517, National Academy of Sciences, National Research Council, Washington, D. C., 1958. Price $2.

A PRIMER OF CEREBRAL PALSY, by *Joseph D. Russ,* M. B., M. D., F. A. A. P., F. A. A. C. P., and *Hyman R. Soboloff,* B. A., M. B., M. D., F. A. A. O. S., F. A. A. C. P. Publication No. 329, American Lecture Series, A Monograph in American Lectures in Pediatrics, edited by *John A. Anderson,* M. D. 77 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $4.

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COMPLETE DENTURE PROSTHESIS, by *Daniel H. Gehl,* D. D. S., and *O. M. Dresen,* D. D. S. Formerly by *Rudolph O. Schlosser,* D. D. S., F. A. C. D. 4th edition. 542 pages; with 310 figures. W. B. Saunders Company, Philadelphia, Pa., 1958. Price $11.

FRACTURES AND OTHER INJURIES, by the *Members of the Fracture Clinic of the Massachusetts General Hospital* and of the *Faculty of the Harvard Medical School,* edited by *Edwin F. Cave,* M. D. Drawings by *Muriel McLatchie Miller.* Photography by *Donald Withee.* Editorial Board: *Joseph S. Barr,* M. D., *Bradford Cannon,* M. D., *Claude E. Welch,* M. D., *James C. White,* M. D., *Edwin F. Cave,* M. D. 863 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $28.

MOULD FUNGI AND BRONCHIAL ASTHMA, A Mycological and Clinical Study, by *P. J. Van Der Werff.* Volume I. With a foreword by Prof. Dr. *Johanna Westerdijk.* 213 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $7.50.

DIAGNOSTIC LABORATORY HEMATOLOGY, by *George E. Cartwright,* M. D. 2d edition, revised and enlarged. 250 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $6.50.

ADVANCES IN ELECTROCARDIOGRAPHY, edited by *Charles E. Kossmann,* B. S., M. D., Med. Sc. D., F. A. C. P. 280 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $6.75.

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ALCOHOLISM, by *Arnold Z. Pfeffer,* M. D. Foreword by *S. C. Franco,* M. D., F. A. C. P. 98 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $6.50.

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